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OF

OPHTHALMOLOGY

EDITED IN ENGLISH AND GERMAN

BY

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OF NEW YORK

AND

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CONTENTS OF VOLUME XXIV.

NUMBER I.

PAGE

1. Strabismus Theories. By Prof. Edward Hansen-Grut, of Copenhagen. Translated and much abbreviated by Dr. J. A. Spalding, Portland, Me. 1
2. The Results of the Operation for Strabismus. By Prof. C. Schweigger, of Berlin. Translated by Dr. J. A. Spalding, Portland, Me. 8
3. Tuberculous Infection of the Eye. By Dr. Ludwig Bach. Translated by Dr. Robert L. Randolph, of Baltimore, Md. (With Plate II. of vol. xxviii., German Edition) 43
4. Oxycephaly and Disease of the Optic Nerve. By Dr. L. Weiss and Dr. O. Brugger. Translated by Dr. Robert L. Randolph, of Baltimore, Md. (With Plate III. of vol. xxviii., German Edition) 55
5. Hyaline and Amyloid Degeneration with Calcareous Degeneration and Ossification in an Ocular Muscle after an Injury. By Dr. Oscar Brugger. From the Eye-Clinic of Dr. L. Weiss. Translated by Dr. C. Zimmermann, Milwaukee, Wis. (With two lithographic plates, XV. and XVI. of vol. xxviii. of German Edition) 65
6. Microphthalmus with Orbito-Palpebral Cyst. By Dr. H. Becker. Abstract from *Arch. f. Augenh.*, vol. xxviii., p. 81, etc., by Dr. Ward A. Holden. (With Plate IV., vol. xxviii., German Edition) 73
7. Tuberculosis of the Lachrymal Gland. By Dr. K. L. Baas. Abstract from *Archiv f. Augenheilk.*, xxviii., 1, by A. Duane, M.D. (With Plate V., of vol. xxviii., German Edition) . . . 76
8. The Changes in the Ciliary Body Following Puncture of the Anterior Chamber. A Contribution to the Study of the Interchange of Fluids in the Eye, and the Formation of Fibrin in the Aqueous Humor. By Dr. Richard Greeff, Privat-docent, Berlin. Translated (abridged) by Dr. Ward A. Holden. (With Plates VII. and VIII., vol. xxviii., German Edition) 81
9. Clinical Contributions. By Dr. J. A. Spalding, Portland, Me. (With a plate in the text) 85
    I.—Empyema of the Orbit Due to Extension of Disease from a Necrosis of the Alveolar Processes of the Superior Maxilla Originally Excited by Exposure to Phosphorus Fumes in a Match Factory.
    II.—A Case of Unilateral Œdema of both Eye-lids with Exophthalmos and Partial Optic Atrophy Persisting Unaltered for Thirty Years.

111 71

PAGE

10. On Ocular Affections in Syphilis of the Brain, with Report of Five Cases. By Charles Zimmermann, M.D., Milwaukee, Wis. . 29

11. Permanent Central Scotoma Caused by Looking at the Sun during an Eclipse, and Complicated by Uniocular, Transient, Revolving Hemianopsia. From Dr. Knapp's Practice, Reported by Dr. A. Duane, New York 104

12. Systematic Report on the Progress of Ophthalmology in the Second Quarter of the Year 1894. By Dr. St. Bernheimer, in Vienna; Prof. C. Horstmann, in Berlin; and Dr. P. Silex, in Berlin. With the assistance of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others. Translated by Dr. Ward A. Holden 109

A.—Dr. St. Bernheimer. General ophthalmological literature, 109; General pathology, diagnosis, and therapy, 110; Instruments and remedies, 112; Anatomy, 114; Physiology, 115.

B.—Prof. C. Horstmann. Anomalies of refraction and accommodation, 116; Lids, 118; Lachrymal apparatus, 118; Muscles and nerves, 118; Orbit and neighboring cavities, 121; Conjunctiva, cornea, sclera, anterior chamber, 123.

C.—Dr. P. Silex. Iris, 125; Choroid, 126; Glaucoma, 127; Lens, 128; Retina and functional disturbances, 129; Optic nerve, 132; Injuries, foreign bodies (parasites), 134; Ocular disturbances in general affections, 136.

13. Miscellaneous Notes 139
Book Reviews.
Appointments.
Prize Essay.
Obituary.
Meetings of Societies.

14. Contents of the Last Number of the *Archiv für Augenheilkunde* (German Edition of these ARCHIVES) 145

NUMBER 2.

1. On the Probable Hemorrhagic Origin of the Striated Affections of the Retina. By Dr. Ward A. Holden, New York. (With two illustrations) 147

2. The Lens-Star Figure of Man and the Vertebrates. By Percy Fridenberg, M.D. (With 21 figures on a plate in the text) . 154

3. A Tumor of the Ciliary Body; with Remarks. By Dr. Mitvalsky. Translated by Dr. Ward A. Holden. (With Plate VI. of Vol. XXVIII., German Edition) 161

4. The Anterior Outflow Channels of the Eye and the Artificial Production of Glaucoma. By M. Knies. Translated and abridged by Dr. A. Duane. (With Plate IX. of Vol. XXVIII., German Edition) 169

PAGE

5. Contributions to the Congenital Anomalies of the Fundus Oculi. By Dr. Mitvalsky, of Prague. Translated (abridged) by Dr. J. A. Spalding, Portland, Maine. (With two chromo-lithographic plates, X. and XI. of Vol. XXVIII., German Edition) . . . 192

6. A Contribution to the Occurrence and Origin of Congenital Anomalies of the Eyes. By Dr. Hugo Wintersteiner, of Vienna. Translated (abridged) by Dr. J. A. Spalding, Portland, Maine. (With one chromo-lithographic plate, XII. of Vol. XXVIII. of German Edition) . . . 197

7. A Case of Double Coloboma of the Choroid. By C. A. Veasey, M.D., Philadelphia, Penn. (With an ophthalmoscopic drawing in the text) . . . 202

8. Diabetic Retinitis. By Oscar Dodd, M.D. . . . 206

9. Alveolar Fibro-Sarcoma of the Optic Nerve. By C. E. Finlay, M.D., of Havana. (With five figures in the text) . . . 224

10. Traumatic Paralysis of the Abducens Nerve. By C. Zimmermann, M.D., Milwaukee, Wis. . . . 243

11. A Case of Anomalous Diplopia, Divergent Strabismus with Homonomous Diplopia; "Two Fixation-Spots" in the Same Eye. By N. Darrell Harvey, M.D., Providence, R. I. . . . 246

12. Congenital Monolateral Fistula of the Lachrymal Sac, Situated Above and Parallel with the Inferior Canaliculus. By John Dunn, M.D., Richmond, Va. . . . 251

13. A Second Attack of Papillitis Occurring in a Case of Post-Neuritic Atrophy of the Optic Nerves. By G. E. de Schweinitz, M.D., and A. G. Thomson, M.D., of Philadelphia . . . 252

14. The Parallax Test for Heterophoria. By Dr. A. Duane, New York, 258

15. Systematic Report on the Progress of Ophthalmology in the Third Quarter of the Year 1894. By Dr. St. Bernheimer, in Vienna; Prof. C. Horstmann in Berlin; and Dr. P. Silex, in Berlin. With the assistance of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others. Translated by Dr. Ward A. Holden . . . 263

A.—Dr. St. Bernheimer. General ophthalmological literature, 263; General pathology, diagnosis, and therapy, 265; Instruments and remedies, 266; Anatomy, 268; Physiology, 268.

B.—Dr. Horstmann. Anomalies of refraction and accommodation, 271; Lids, 273; Lachrymal apparatus, 274; Muscles and nerves, 275; Orbit and neighboring cavities, 277; Conjunctiva, cornea, anterior chamber, and sclera, 280.

C.—Dr. Silex. Iris, 285; Choroid, 286; Glaucoma, 287; Sympathetic ophthalmia, 287; Vitreous, 288; Lens, 288; Retina and functional disturbances, 290; Optic nerve, 293; Injuries, foreign bodies (parasites), 294; Ocular disturbances in general affections, 295.

PAGE

16. Miscellaneous Notes 299
    Appointments.
    Meetings of Societies.

17. Contents of the last number of the *Archiv für Augenheilkunde* (German Edition of these ARCHIVES) 299
    A.—Original Papers.
    B.—Report on the Progress of Ophthalmology in the Third Quarter of the Year 1894. (Translated in this number).

NUMBER 3.

1. Observations on the Macula Lutea. By G. Lindsay Johnson, M.A., M.D., Cantab., F.R.C.S., London. (With ten microphotographs in the text) 301

2. Congenital Night-Blindness and Pigmentary Degeneration. By Dr. Colman W. Cutler. (With two lithographic plates, I. and II., of Vol. XXX., German Edition) 313

3. Three Unusual Cases of Retino-Choroidal Degeneration. Reported by Colman W. Cutler, M.D. (With drawings by Dr. Salzmann. Pl. III., Vol. XXX., German Edition) 334

4. A Straight Arm, Corrected Scale, and other Improvements for Javal's Ophthalmometer. By Carl Weiland, M.D. (With three drawings in the text) 340

5. How Javal's Keratometer may be Easily Changed into a Good Chromatometer for the Examination of Patients as to Color-Blindness. By Carl Weiland, M.D. 349

6. A Case Illustrating the Bad Effects which the Establishment of Menstruation may have upon the Course of Interstitial Keratitis. By John Dunn, M.D., Richmond, Va. 353

7. A Case of Dermoid Cyst of the Orbit. By B. L. Millikin, M.D. . 356

8. Traumatic Paralysis of the Abducens Nerve. By H. Moulton. M.D., Ft. Smith, Ark. 363

9. Diplopia Occurring at the Periphery of the Field of Fixation. By Dr. A. N. Alling, New Haven, Ct. (With three figures) . . 365

10. A New Method of Tying the Sutures in Advancement of the Ocular Muscles. By G. Melville Black, M.D., Denver, Col. (With four figures) 375

11. A Clinical Study of One Hundred and Sixty-seven Cases of Glaucoma Simplex. By William Zentmayer, M.D., and William Campbell Posey, M.D. 378

12. An Unusual Case of Hyaline Bodies in the Optic Nerve. By Dr. H. Gifford, Omaha, Neb. (With a drawing in the text) . . 395

PAGE

13. An Orbicularis Pupillary Reaction. By Dr. H. Gifford, Omaha, Nebraska 402

14. Treatment of Syphilitic Affections of the Eye by Mercury, Potassium Iodide, and Pilocarpine Combined. By G. Herbert Burnham, M.D., F.R.C.S. Edin., M.R.C.S. Eng. 407

15. Systematic Report on the Progress of Ophthalmology in the Fourth Quarter of the Year 1894. By Dr. St. Bernheimer, in Vienna; Prof. C. Horstmann, in Berlin; and Dr. P. Silex, in Berlin. With the Assistance of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others. Translated by Dr. Ward A. Holden 410

A.—Dr. St. Bernheimer. General ophthalmological literature, 410; General pathology, diagnosis, and therapy, 411; Instruments and remedies, 412; Anatomy, 414; Physiology, 415.

B.—Dr. C. Horstmann. Anomalies of refraction and accommodation, 417; Lids, 418; Lachrymal apparatus, 420; Muscles and nerves, 421; Orbit and neighboring cavities, 422; Conjunctiva, cornea, sclera, and anterior chamber, 425.

C.—Dr. P. Silex, Iris, 428; Choroid, 430; Glaucoma, 431; Sympathetic ophthalmia, 432; Lens, 432; Vitreous, 434; Retina and functional disturbances, 434; Optic nerve, 438; Injuries, foreign bodies (parasites), 439; Ocular affections in general diseases, 440.

16. Obituary 444
Dr. M. Landesberg.

17. Book Notice 444
Skiascopy and its Practical Application to the Study of Refraction. By Edward Jackson, A.M., M.D.

18. Contents of the Last Numbers of the *Archiv für Augenheilkunde* (German Edition of these ARCHIVES) 444

NUMBER 4.

1. Observations on Cases of Hemichromatopsia, Indicating the Non-existence of a Separate Cortical Color Centre. By Dr. Ward A. Holden 447

2. On Choroidal Tumors. By Dr. Mitvalsky, Prague, Austria. Translated and abridged by Dr. Ward A. Holden. (With seven figures on Plates XVIII. and XIX. of Vol. XXVIII. German Edition) 450

3. Contributions to the Knowledge of the Histological Alterations in the Retina after Experimental Injuries. By Dr. A. Tepljaschin, of Kasan, Russia. Translated (abridged) by Dr. Ward A. Holden. (With figs. 1–28, Plate XXI., XXII., and XXIII., Vol. XXVIII., German Edition) 455

4. The Palpebral Form of Spring-Catarrh. By Dr. H. Gradle, Chicago 466

5. A Case of Diphtheritic Conjunctivitis Presenting Unusual Complications Treated by Antitoxin. By Dr. J. Guttman, New York . 472

PAGE

6. A Dermoid Tumor Involving the Cornea, with Remarks. By L. D. Brose, M.D., Ph.D. 479
7. A Case of Successful Extraction of a Piece of Steel from an Iris in which a Plastic Inflammation had been Established, with Prompt Subsidence of the Inflammatory Reaction and Restoration of Full Vision. By Edward B. Heckel, A.M., M.D. 482
8. Report and Experiences of an Itinerant Cataract Operator at the End of the Nineteenth Century. By Dr. O. Walter, Odessa, Russia. Abridged and translated by H. Knapp 484
9. Light—the Ointment of the Yellow Oxide of Mercury—and the Ointment-Pots in Common Use. By S. Holth, of Drammen, Norway. Translated by Charles H. May, M.D., New York . 486
10. Diplopia in the Periphery of the Field of Fixation and its Bearing on the Diagnosis of Muscular Paralysis. By Dr. A. Duane . 491
11. Report of the Transactions of the Twenty-fourth Ophthalmological Congress, Held in Heidelberg, Aug. 4 to 7, 1895. By Prof. Horstmann and Dr. Greeff, Berlin. Translated by Dr. C. Zimmermann, Milwaukee, Wis. 498
12. Systematic Report on the Progress of Ophthalmology in the First Quarter of the Year 1895. By Dr. St. Bernheimer, in Vienna; Prof. C. Horstmann, in Berlin; and Dr. P. Silex, in Berlin; with the assistance of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Dr. Herrnheiser, Prague; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. R. Greeff, Berlin; Dr. Deus, Berlin; and others. Translated by Dr. Ward A. Holden 513
    A.—Dr. St. Bernheimer. General ophthalmological literature, 513; General pathology, diagnosis, and therapeutics, 516; Instruments and remedies, 516; Anatomy, 518; Physiology, 519
    B.—Dr. C. Horstmann. Anomalies of refraction and accommodation, 521; Lids, 522; Lachrymal apparatus, 524; Muscles and nerves, 524; Orbit and neighboring cavities, 526; Conjunctiva, cornea, sclera, anterior chamber, 528.
    C.—Dr. P. Silex. Iris, 531; Choroid, 532; Glaucoma, 532; Sympathetic ophthalmia, 533; Lens, 533; Retina and functional disturbances, 537; Optic nerve, 542; Injuries, foreign bodies, parasites, 543; Ocular disturbances in general affections, 545.
13. Book Reviews 549
    Perimetry. By Dr. Ole Bull.
    Vorlesungen über den Gebrauch des Augenspiegels (Manual of Ophthalmoscopy). By Prof. C. Schweigger and R. Greeff.
14. Obituary 555
    I.—Henry Willard Williams, A.M., M.D.
    II.—Dr. Albrecht Nagle.
    III.—Dr. Vialet.
    IV.—Dr. T. Inouye.
15. Appointments and Honors 557
16. Contents of the Last Number of the *Arch. f. Aug.* (German Edition of these ARCHIVES), Vol. xxxi., No. 2 557
17. Index 559

VOL. XXIV. No. 1

ARCHIVES OF OPHTHALMOLOGY.

STRABISMUS THEORIES.

BY PROF. EDWARD HANSEN-GRUT OF COPENHAGEN.

Translated and much abbreviated by Dr. J. A. SPALDING, Portland, Me.

PROF. WAHLFORS has greatly honored me by subjecting to a detailed critique[1] my theory of strabismus, in which he fails to see anything at all correct.

All strabismus theories, Schweigger's, Stilling's, Wahlfors', and my own, for instance, are unsubstantial, of course, but Prof. Wahlfors' summary of my theory is so incorrect, and his own is so false, that I must ask an opportunity to give in these ARCHIVES a brief *résumé* of my actual train of thought.

In order to discover the origin of strabismus, it is important to know the actual position which the eyes occupy when at rest, *i. e.*, the anatomical position of rest resulting from the form of the orbit, the insertion of the optic nerve, and the natural length of the muscles when not innervated. Many things go to show that this position is more or less divergent, rarely parallel, and hardly ever convergent. This *anatomical* position of rest is the starting-point of every co-ordinate action of the interni ; it is the absolute zero point of convergence. The function of the eyes never allows divergence. Hence the parallel direction demanded for distance can never be abandoned whilst we are awake, and the innervation of convergence, which pulls the eyes from the anatomical position of rest to parallelism, is simply maintained by habit, by which expression I mean *unconscious innervation.* Therefore parallelism is the position which we find exclusively in eyes with normal refraction and binocular vision. This, then, is the *functional* position of rest,—functional because the function of the eyes has produced it, and position of rest, because the position assumed by habit and unconscious innervation is free from all exertion. This also is different from the anatomical position of rest, (which likewise is free from innervation,) and the amplitude of convergence which lies between the anatomical and the functional point of rest is latent.

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[1] *Archiv f. Augenheilkde.*, vol., xxvii., Nos. 3 and 4.

Now more than this, the relation of convergence to accommodation in reference to the functional position of rest plays a rôle, in that in Hm we oftener see a convergence under the covering hand than in Em, whilst in M we observe a divergence. I agree with Schweigger that in a majority of cases of Hm, the position is correct, or nearly so, in fixation at all distances, which shows that the capacity of suiting the accommodation and the convergence to the distance is easily learned. This now presupposes that the convergence must be separated from the accommodation, for, even if we do not consider as the only normal condition the exact agreement between accommodation and convergence which we observe in Em, an equal innervation when the two muscles (interni and ciliary) always act together is the most natural condition. It is therefore probable that no variation from this condition can exist without a certain amount of practice.

When we order glasses for Hm or M, it is the rule that the correcting lens is well endured for all distances, though there are cases in which the glass for distance is too strong for reading. So, too, we find the correct position in most cases of Hm behind the covering hand, and, in addition, cases of latent convergence which are more frequent than in other refractive conditions. But that this convergence is not the anatomical position of rest is shown by its disappearance after wearing correcting lenses.

Such a latent convergence produced by Hm is the beginning of strabismus, and passes into manifest strabismus when the usual adjuvantia are present.

I further believe that habit (unconscious innervation) maintains parallelism under the covering hand even with a divergent anatomical position of rest. So, too, in a few hypermetropic eyes without binocular vision, in which convergence cannot free itself from the accommodation, a convergent position is the habitual one, *i. e.*, a *permanent squint.* In other words, constant or exaggerated convergence, without binocular vision, finally robs the individual of his capacity of entirely abandoning his convergence innervation, and the new position produced by the function is assumed instead of the anatomical point of rest. In addition to this functional permanent squint, accommodation in acute fixation produces increased convergence (accommodative increase of strabismus).

Permanent convergent strabismus is therefore an inward movement of the functional position of rest, and whether this produces convergence, or parallelism, or but slightly decreased deviation (starting from the anatomical point of rest), depends on the amount of the divergence of the latter. With slight divergence, habitually strong convergence will produce permanent convergent squint; with greater divergence, the convergence results in parallelism. Hence, when lost in thought we have functional position of rest or parallelism; during fixation, a slight or greater convergence,—in other words, periodic strabismus.

If the anatomical position of rest is still more divergent, the habit of convergence may produce an inward movement of the functional position of rest, but it never reaches parallelism. Hence we have, during fixation, accommodative convergent strabismus; when lost in thought, divergence.

I therefore think that unconscious habitual innervation, which causes permanent convergent squint, is maintained even when the accommodation is not active.

The great importance of habit in inducing various innervated positions *without conscious exertion* is observed in many occupations in which fixed positions of the whole or of various parts of the body are frequently and uninterruptedly assumed as the most suitable of all.

Uninterrupted innervation is something which my critic cannot understand, and yet he has the same thing in mind when he says: "We cannot always rely on the equilibrium test giving a correct solution, because the habit of keeping the eyes parallel is so great that, though binocular vision is lost, it maintains the eyes in this position, when the deviation is of no great consequence." I ask, what else is this "*habit*" than an unconscious innervation which keeps the eyes parallel, although the position of rest is not itself parallel !

Physiology also teaches such an unconscious innervation. The physiologists do not grant that the muscles are ever free from innervation. They have a "tonus," which is defined as an unconscious innervation to contraction, and I should not object if it were generally agreed that the inward movement of the functional position of rest, as suggested by myself, is simply an increased tonus.

Divergent strabismus is, in my opinion, directly opposite to convergent, being not so much an opposite form of deviation as a partial cessation of the innervation of convergence. *Convergent squint is the result of an actual innervation of the interni : divergent squint is a passive process.*

Wahlfors' theory depends on *the position of equilibrium*, by which he understands the position which the eyes would take if the muscles were free from innervation, or from the elastic tension of the muscles. He imagines the eye suspended amidst so many elastic bands. If one muscle is more powerfully developed than another, it answers with a greater contraction, and the harmony between the muscular movements is disturbed. But this talk of stronger and weaker muscles is, in my opinion, a mere figure of speech.

"Normal muscles," says Professor Wahlfors, "give a parallel position of equilibrium, but its *actual* position is not always definable, because *habit* maintains the parallel position. We can be sure that it is parallel when the muscles are normal."

All this is arguing in a circle. How does he decide that the muscles are normal? By the greatest convergence and simultaneous abduction which they can produce. But this only proves the *innervation*, *not the muscular strength.* Whilst each eye can move out about 40°, there is no innervation of the simultaneous abduction of both eyes, and it does not seem as if we could estimate the strength of the externi by mere *lack of motion.*

Then, again, the manner in which Professor Wahlfors establishes the near convergence point is no proof of the muscular strength. He simply lets the person look at a small object, but many a myope will let one eye deviate early, in such a test, because the maintenance of binocular vision would prevent *distinct vision.*

So far as simultaneous abduction is concerned, we can reply that it is absent, because the individual possesses the power of so-called facultative abduction.[1] This averages a prism of about 6°, and is very minute in comparison with abduction in the service of lateral movements. This difference depends on a variation in *innervation, and has nothing to do with the* strength of the muscles. Hence it is absurd to conclude from a variation of about 2° to 3° of prismatic effect of facultative abduction, that in one case the muscles are strong, and in another weak.

It is amazing to see our best authorities write about "weakness of the externi, or exaggerated strength of the interni," and yet it is plain that unilateral weakness of one muscle would produce the same symptoms as unilateral muscular paralysis! No matter where the defect lies—in the gray substance, or ganglia, or nerve tracts, or in the muscles themselves,—the muscle, in order to act under such circumstances, in order to do the same work as a normal muscle, must have an additional nerve impulse. But here we reach the subject of paralytic strabismus, and leave concomitant strabismus behind.

I have tested thousands of people for equilibrium, partly with a screen and partly with a vertical prism. Variations equal to a prism of 1° or 1.50° I have regarded as parallel, for they cannot be objectively proved, nor do I believe that they can be utilized in founding a theory for strabismus. As the deviation is generally convergent in Em and Hm, and divergent in M, I feel inclined to regard it as the functional position of rest. Larger deviations in a divergent direction were not common, whilst the same in a convergent direction were excessively rare, always seen in Hm, and disappearing on using glasses. Similar convergence is rarely seen in Em, and possibly a little more often in M.

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[1] Our knowledge of the physiology of the eye muscles is defective in many points important to our knowlege of strabismus. Is, *e.g.*, facultative abduction an expression of simultaneous contraction of the externi, or the extreme relaxation of convergence innervation? Is it an expression of an active or of a passive condition?

These results vary greatly from those of Stilling, who found equilibrium in most cases of Hm and Em, and divergence in M. I attribute this difference in our results to the fact that Stilling picked his patients from those who could read with ease.

Stilling's theory is that most people squint, and that it is only our great power of fusion which prevents strabismus from being more often manifest.

Wahlfors and Schweigger employ the argument which asserts that Hm is the cause of convergent squint because two thirds of all the cases of this nature have Hm. The other third have another causation, which is correct, because Hm is not the only cause of excessive convergence, and we have the right to ask of these authorities what proof they have that the convergent position of equilibrium is the cause of strabismus. When they see a patient with strabismus, they cannot prove that a convergent position of equilibrium *preceded the squint!*

In the latter part of his paper Wahlfors describes how strabismus originates from various errors of refraction. His idea of the positions of equilibrium would prove that a hypermetrope's *tendency* towards accommodative convergence more easily passes into genuine convergent strabismus, when the position of equilibrium is convergent to star with, than when it is parallel or divergent.

We answer now those views of the importance of the convergent position of equilibrium in the various conditions of refraction, in this way:

(1) The convergent position is not anatomical but functional (innervated). (2) It is the beginning of the squint, not the cause. (3) The existence of the position previous to the squint cannot be proved, but only suspected from experience in certain cases of asthenopia, the significance of which just at present is not precisely apparent.

I am glad to agree with Wahlfors when we approach divergent strabismus, for I too believe that it presupposes a divergent anatomical position of rest. If this is present, the eyes pass into it when *the innervation of convergence ceases.*

Prof. Wahlfors errs in opposing Tscherning, who thinks that the result of a squint operation depends on the retraction of the divided muscle, whilst Wahlfors argues that the effect depends upon the action of the antagonist. This is correct in my opinion also, but Prof. Wahlfors believes that the difference in effect between loosening an internus and an externus depends on the greater adhesion of the externus to Tenon's capsule, which offers greater resistance to convergence of the eye after tenotomy. But the condition of affairs is not so. In convergent strabismus we have not only an *active innervation* of the internus but of all the ocular muscles. The eye is turned outward by the actively innervated externus after dividing the internus. In divergent squint, on the contrary, which depends on complete relaxation of the convergence innervation, the action of dividing an externus is relatively slight, because the antagonist, the internus, *is not innervated at all!* And it is in this difference between the action of tenotomy of an externus and of an internus that I find support for my *innervation theory of strabismus.*

My chief idea in writing this paper has been to correct criticisms of my views on strabismus, and in order to be brief I have been obliged to omit the opinions of many authorities. Careful study of the whole question of the pathology of strabismus will prove to any one that many points in this province are still doubtful to us all. Nevertheless I believe that by following the innervation theory as the cause of the positions of equilibrium so variously understood, we are on the path leading to the explanation of many still unanswered questions.

THE RESULTS OF THE OPERATION FOR STRABISMUS.

By Prof. C. SCHWEIGGER, of Berlin.

Translated by Dr. J. A. Spalding, Portland, Me.

THE operation for strabismus is an every-day occurrence in the practice of a busy ophthalmic surgeon, but it is curious, that we nowhere find any account of the results obtained. The results used to be measured by the amount of retrogression of the squinting muscle, generally estimated from 3 to 5 *mm*, though the measurements were not very accurate. Tenotomy, and the consequent retrogression of the faulty muscle is the first, but not the only deciding point in estimating the final result of the operation.

The cases best adapted to determine the success of a simple tenotomy are those in which diplopia is present, because from the distance between the images we can easily decide the amount of deviation before and after the operation by tests that are familiar to all. It would be a most desirable thing if we could fix the deviation from parallelism, but, to speak accurately, proper space for such a test is rarely at command, So far as the determination of refraction is concerned, 5 to 6 *m* are quite sufficient to regard as infinity, but this distance will not do in considering questions of equilibrium, because the distance between the eyes, amounting to 64 *mm*, cannot be regarded as zero at any such distance as 6 *m*. If we determine the weakest prism that unites the two images of a candle, we do not in the act exclude all muscular energy, for at a certain degree of nearness, double images will be fused by innervation in the interest of binocular vision.

So, too, where binocular single vision for distance still exists, diplopia can often be produced by a red glass before one eye. The distance between the images will then be fixed by the prism that unites them. The influence of binocular vision is excluded in tests for equilibrium, and the prism which brings the images above one another in the vertical test measures the deviation. And here I insist that in all these tests we have to rely on what the patient says, and that we have no means of verifying his statements, so that our opinions may be incorrectly influenced by his assertions. Then again we must bear in mind that we are testing living muscles, and personal experience must tell us that the tension and capacity of these vary largely. Hence repeated examination may bring different replies. Nevertheless, despite this source of error, it will be satisfactory if we regard the squinting deviation, measured by prisms, as half of the refracting angle. If we find prisms stronger than 18° needed to fuse the double images, they must be divided between the eyes.

Simple tenotomy is performed as follows: The conjunctiva over the insertion of the muscle is seized with the forceps, and a small incision made, into which the forceps are introduced, the tendon grasped, and opened close to its insertion. Through the orifice I next pass a small blunt hook, stretch the tendon and separate it from the sclera with scissors, passing the latter first up and then down, or *vice versa.* I insist on distinctly seeing the insertion of the tendon, and I only consider the separation as perfect when the hook, passed up and down behind both ends, can pass freely to the corneal margin. It may happen, especially in tenotomy of the internus, that above or below the insertion the hook meets with an obstruction which has nothing to do with the muscle, but is the anterior venous stem from the choroid, the division of which may be followed with copious hemorrhage, but otherwise is of no account. If we desire an extensive effect the scissors, after making the conjunctival incision, are pushed along the anterior surface of the muscle to the caruncle, and the tissues loosened freely to divide the fibres that pass from the internus to the caruncle, and to let the muscle retract as much as possible, as suggested by

Liebreich.[1] We should not forget that in those days it was thought improper to loosen the muscle from the conjunctiva in this operation. If the conjunctival incision is too large, a suture is inserted, but not too deeply, because if applied horizontally so as to enter the subconjunctival tissue in Tenon's capsule, it may spoil the result of the tenotomy by drawing forward the divided muscle. Hence we must only suture the actual mucous tissue, and do it vertically. If the conjunctival incision is small a suture is superfluous, and I have never seen any harm from omitting it. The idea that it is necessary to prevent the sinking of the caruncle is overdrawn. The best means to obviate this occurrence is to divide the fibres passing from the internus to the caruncle.

SIMPLE TENOTOMY OF THE INTERNUS.

I have operated but twenty-five times on convergent squint with normal binocular vision, the result of which could be measured after the operation. Of these, two had Em, three had Hm, thirteen M, one Ahm, and six were anisometropic, two being Em and Hm and four Em and M.

The squint in all of these patients developed at an age when binocular vision had become a habit. The youngest was six years old, the oldest forty. Diplopia was present in all. The double images deviated more in the boundaries of the field to the right and left than in the median lines, unless abducens paralysis was present. Only eleven patients remained long enough to enable us to get a correct idea of the result of the operation. Owing to the rarity of such observations, I will describe some in brief.

Case 1.—Miss A., age seventeen, April, 1879, periodic squint O S, for six months, with diplopia. M : O U, about — 3. With proper lenses and red glass, diplopia at 5 M corrected by prism 16°. In May tenotomy of internus O S. In two weeks diplopia by vertical prism, corrected by prism 16° base out. Later still diplopia as before, with periodic convergence. In July tenotomy of internus O D. Result good. Three years later occasional diplopia. Next year, no diplopia; no facultative divergence. Homonymous diplopia with red glass test.

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[1] v. Graefe's *Archiv*, xii., 2, 298.

In 1893, fourteen years from first test, prism of 14° to correct red glass diplopia. No objective diplopia. Occasional periodical squint.

Result: Tenotomy on squinting eye at first satisfactory; later less; later still, tenotomy on fixing eye; at first satisfactory; did not remain so.

CASE 2.—Mr. H., age thirty-two, recent diplopia July, 1877. M 6 O U, fixes with O D. With correction of M, and vertical prism, doubled images corrected with prism 40°. Tenotomy internus O S. Correction of vertical-test diplopia with prism 9°. Diplopia persists in October, but less. The result being nil, tenotomy was done on internus of O D, with subconjunctival loosening to the caruncle; result, a permanent cure at the end of two months.

Here, too, tenotomy on the squinting eye was of no use; success was only attained by operating on fixing eye.

CASE 3.—Miss K., age twenty-four, convergent squint for six years, mostly in O D. Diplopia frequent. M: O D — 9, O S — 8. With correction of M, vertical test-images united with prism 24°. November, 1884, tenotomy of internus O D. Good result. Artificial diplopia corrected with prism 14°. In July following, red glass diplopia; same prism. No diplopia without red glass.

The apparent correction was not permanent. Four years later convergence as before. O D also lower than O S, the vertical deviation being corrected with prism 7°, and the lateral (in vertical tests) by prism 28°. Tenotomy of internus O S. Result fair; *lateral deviation less, but vertical greater.* On fixing with O D, secondary deviation downward greater than upward in other eye. In six months the latter was cured. In 1891 position of eyes good. Binocular fixation.

The remarkable feature in this case is the increased vertical deviation after tenotomy of internus O S. That it was not artificial was proved by its gradual disappearance.

CASE 4.—Miss G., age eighteen. Homonymous diplopia for some years. Image of O S higher; deviation increasing on looking to left. Defective mobility of O S to left, but not more than usual in such cases. No reason to presume abducens paralysis. Personally I have never seen abducens paralysis followed by periodic squint. Prism of 14° fuses lateral images. October, 1888, tenotomy of internus O S. No subconjunctival loosening or suture. A week later diplopia fused by prism 7°. Second week prism of 12° needed. The result being unsuccessful, internus of O D tenotomized, after which no diplopia.

Here is another instance of squint only disappearing after tenotomy in the fixing eye.

CASE 5.—Miss E., age fifteen. M: O U. Periodic convergent squint O D, with diplopia. Tests show prism of 34° to correct. June, tenotomy of squinting internus. Prism of 26°, then of 10°, later on 4°, finally relapse, with facultative divergence. Ultimately, tenotomy on fixing eye with permanent good result.

CASE 6.—Miss F., age twenty-nine. Periodic convergent squint O S, one year. M — 5 D S ; O U. Homonymous diplopia corrected by prism 38°. Tenotomy of internus O S, resulting in prism of only 7°, but later, relapse and prism of 28° needed to fuse. Tenotomy on the squinting eye was here again of no use.

CASE 7.—Louise, age eight. Diplopia for two years. Hm. O U + 1 D S. Prism of 34° to correct vertically placed images. March, 1893, tenotomy of internus O D. A week later, image of O D lower than that of O S. In May, binocular vision. Prism of 16° to fuse artificial diplopia. In July, prism of 8°. No red glass diplopia.

This is the first case in which I saw gradual increase in the effect of a tenotomy on the squinting eye, amounting after a year to a prism of about 26°, *i. e.*, 13° angular measurement.

CASE 8.—Fritz, age sixteen. Periodic convergent squint O S ; M, and Am. — 5 D S O U with — 1.5 D C. Diplopia by red glass corrected by 18° prism, and proper lenses.

May, 1893, tenotomy of internus O S. June: prism 2° corrects red glass diplopia. No facultative divergence. The result of tenotomy on squinting eye was consequently a prism of 18°.

CASE 9.—Mr. B., æt. forty. 1892. Recent diplopia, M : O U about — 5 D S. Convergent squint O S. Defective motion outward O U. Left image lower than right. Red glass diplopia fused with prism 30°. October: Tenotomy of internus of squinting eye. Correction with 5° prism. At end of three weeks diplopia worse. Symptoms of abducens paralysis ensued, and remained till following January. By May no diplopia, no paralysis. Vertical-test diplopia fused with 15° prism.

The result of the operation, after disappearance of intercurrent abducens paralysis, amounted to prism of 10°, and binocular vision was maintained.

CASE 10 shows peculiar complication with abducens paralysis. Miss S., September, 1892, consulted me for abducens paralysis O D. *Nevertheless patient fixes with O D, and has convergent squint of O S!!*

April, 1893, diplopia increased by looking to left. Vertical-test images corrected by prism of 38°. Tenotomy internus O S. Vertical conjunctival suture. Result, nil.

The direct result of the tenotomy could be determined in only twenty of these twenty-five patients with normal binocular vision and measurable deviation of the double images, and the final result in only ten. We soon discover a poor result. For that reason I have taken cases of long standing, the shortest being six weeks, and the longest being several years. I feel that we ought not to measure the prismatic deviation, but the angular, because we then obtain results which sufficiently express the correction of the visual angle. The immediate result averaged 8°. Still this is not the final result of tenotomy on the squinting eye, which was satisfactory in but three cases out of ten. The average was 8°. Case 10 was not seen again.

A satisfactory and permanent result was obtained by operating on the fixing eye in two cases, the result averaging 10°. Cases 5 and 10 obtained a direct result by tenotomy on the squinting eye corresponding to a visual angle of 14°, but soon disappearing. At first thought it would seem as if cicatrization were at fault, in relapses, but this cannot be so always, for in Cases 6 and 9 tenotomy on the squinting eye gave a permanent result. So in Case 5, tenotomy on the fixing eye gave permanent result of nearly 16°. Cicatrization, therefore, has not much to do with failures, and the cause must lie deeper. In the same way these cases show a defect in the theory of mere relaxation of the tendon.

STRABISMUS WITHOUT DIPLOPIA.

This group differs from ordinary squint in the persistence of normal binocular vision. The distinction is important physiologically, for the success of the operation, though not so much so as would seem at first glance. It is true that the eye muscles are controlled by the persistence of binocular vision, so that a defective result in the operation is compensated for by increased innervation in the interest of binocular vision. But if, as happens in most cases of squint, binocular vision no longer exists, a defective position of the squinting eye does not disturb the patient, so that nothing remains but the question of the cosmetic effect. Is the eye still noticeably deviated? Here we have to depend on the position of the corneæ. Yet apparent squint shows the deceptions to which we may be exposed, for normal binocular vision may persist and yet the eyes look crossed. The opposite condition, in which the tests show squint but the eyes are straight, is much more common.

Another important point in the estimation of the operative result, is that in most cases of squint the deviation is alternate. Periodic and permanent squint are not distinctly separated. Periodic squint occurs in Hm as well as in Em or M. Many cases of permanent squint begin as periodic. Hence it is not surprising that the amount of deviation is not constant but varies, as can be proved by *repeated* measurements. There may be cases which always have the same deviation, but we can only verify this fact by careful tests.

Squint being rather common, and every case needing accurate definition, we must employ some simple means for its determination.

Horner's suggestion to measure with the perimeter is too minute, for the squinting eye is placed in the centre of the arc and the fixing eye gazes at a distant object. A candle is then passed along the arc till its reflection falls exactly into the middle of the pupil of the squinting eye. The angle marked on the arc gives the angle of squint, though the angle "*a*" should be considered. But who can guarantee that the fixing eye remains steady? Every one knows how patients miss this in perimetrical measurements. Then there is the oblique position of the head in squint; or, again, if the squinting eye fixes the centre of the perimeter, how can we prove that it remains so? It has been proposed to steady the head; but has it been successfully done? The minuteness of the test explains why its supporters will not listen to any variation in the squinting angle. But if this fact can be shown by linear measurements, the imperfections of the perimeter are patent. In this point of view, Landolt[1] is right when he says that the angle of squint does not change from moment to moment, but at longer intervals, as in periodic squint.

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[1] v. Graefe's *Archiv*, xxv., 3, p. 271.

Hirschberg has suggested this simple method.[1]

If we place in the median line of the patient's head a flame at a distance of about a foot, the examiner, who stands behind the light and shades his eyes, will see the reflexes of the flame in the corneæ. If the patient fixes with both eyes, the images will be symmetrical in both pupils. If the image in the squinting eye stands at the margin of a medium-sized pupil, the angle of squint will measure about 13°. If it lies beyond the margin of the pupil, about 25°. Although Landolt[2] considers this method sufficiently accurate, it only shows that he does not lay much stress on measurements, for it is nothing but an off-hand guess.

v. Graefe's method is much more accurate. The best way to perform it is to start with the deviation from parallelism as a base, though we must not take the word parallel too literally, because it is difficult to find a room 50 *m* long. I lay more stress on the fact that the measurement is made at a distance in which the accommodation is about as much at rest as in looking at a distance. If we fix an object at 30 *cm*, the squint is often increased by the accommodation of convergence. Hence I first let a flame be fixed by the squinting eye at a distance of 3 to 5 *m*, and then bring the zero point of the scale beneath the corneal image of the flame. Then the fixing eye is arranged for the same flame, and we measure how far the middle of the pupil of the squinting eye (not the corneal reflex) has deviated from zero. In doing this we must select our position so that we stand in the visual axis of the squinting eye—*i. e.*, in right convergent squint on the left side of the patient, and in right divergent squint on the right side. The secondary deviation of the fixing eye is measured in the same way. The difficult cases are those in which with high degrees of amblyopia the squinting eye does not fix. Here we can assist ourselves by letting the fixing eye deviate till the reflex of the flame falls into the centre of the pupil of the squinting eye.

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[1] *Centrabl. f. Augenheilk.*, vol. x., p. 5.

[2] v. Graefe's *Archiv*, xxv., 3, p. 271.

In order to determine the results of tenotomy of the internus I have tabulated a list of cases in which this operation was done. More than half were of periodic convergent squint, in which I also included patients who squinted nearly all the time, but not constantly. In others, the frequency of the periodicity and its amount were indications for the operation, except that I operated in Hm only when convex lenses failed to relieve the squint. If the relaxation by tenotomy theory is correct, periodical convergent squint ought to be changed by tenotomy into periodic divergent squint, because binocular vision, the physiological regulator of the eyes, is absent. But binocular vision is as easily forgotten in periodic as in permanent squint, and even amblyopia is not rare in these conditions.

Diminution of vision, congenital or not, is one of the causes of permanent as well as of periodic squint. If the latter originates at an age when binocular vision has become a habit, it generally begins with a diplopia, which gradually disappears, and at last double images can only with difficulty be produced, just as in permanent squint. But for all that, the results of the operation are perfect in a vast majority of cases. So, too, it may happen that tenotomy on the squinting eye is useless, as we soon discover. Such cases are rare and cannot be foreseen, and the remedy lies in tenotomy of the fixing eye. It may also happen that periodic squint is rarer after tenotomy, and also less perceptible. *Still, in a majority of cases, periodic squint is removed by simple tenotomy.* The only useful cases are those that remain under observation sufficiently long to enable us to form an opinion in regard to the result. Most of our operated patients disappear as soon as the eye ceases to be red, but I have succeeded in collecting a few cases of periodic squint seen for nearly a year after the operation, and in these we see the variations in the result of simple tenotomy.

Some of these cases had a minute deviation, and others a deviation of *7 mm*, and yet tenotomy had not done too much for the former, or too little for the latter, the explanation being that the maximum of deviation is rare, and that the cases, with but slight deviation, could at other times squint to a greater degree. Nor should we fail to observe, *that the return of periodic squint is generally prevented by simple tenotomy, of which I will here cite an instance.*

Case 11.—Miss A., aged eight, consulted me in 1879 for periodic convergent squint, O D, persisting for two years, and coming on only every other day. On the squinting days the squint was 7 *mm*, with equal secondary deviation of the other eye. Hm + 2 D S O U. But Hm cannot cause squint only every other day! Lenses did not remove the squint on the squinting days. Every other day the eye was absolutely straight. Under the screen slight deviation. Quinine of no effect on periodicity. Homonymous diplopia with red glass and vertical prism, but no fusion with prism. No stereoscopic fusion. In other words, no binocular fusion. Tenotomy of internus O D. A year later, O D constantly straight. Slight convergence behind screen. No binocular vision. Wears correcting lenses for distance, but no squint for near work without lenses.

Result: Instantaneous disappearance of periodic squint of tertian type.

The tertian type is as inexplicable as its disappearance after tenotomy, as is also the case when an occasional squint is relieved by tenotomy.

The first necessity in periodic squint is tenotomy. An imperfect result can be supplemented by tenotomy on the other eye. I often tenotomize both eyes, because many cases must be included in periodic squint, as, for instance, when the squint is occasional, or when the patient does not squint all the time. We also have to consider the maximum of convergence, which may be very great, as in:

Case 12.—Marie, aged six, periodic convergent squint O D for two years. Deviation 6 *mm.* Slight M. Tenotomy of both interni. Good result. Occasional slight divergence on fixation at 30 *cm.* Eleven years later M somewhat increased. Position of eyes good. Light reflex in middle of pupils. Slight convergence behind screen, more marked O D than O S. Occasional homonymous diplopia with red glass and vertical prism.

Case 13.—Anne, aged seven, periodic convergent squint for two years. Maximum, 6 *mm*, Ahm O U. Tenotomy of both interni November, 1880. Good position obtained. No diplopia, butstereoscopic fusion. Result still good five years later.

Case 14.—A. P. Alternating periodic convergence for two years, mostly in O D. Hm O U + 2 D S. Two years later squint persists despite lenses. Maximum 9 *mm.* The minimum could not be measured, as fixation increases the deviation. Transitory diplopia. February, 1880, tenotomy of both interni. Diplopia disappeared. Position good. Convergence when either eye is covered. With lenses, position good all the time; without lenses, marked permanent convergence. *With lenses also, periodic accommodative convergent squint.* Ten years later the same.

The case teaches us that positive accommodative squint could not be corrected by lenses or by double tenotomy.

Periodic squint passes without well defined limits into permanent squint, for in the latter the deviation is not always the same, so that it takes many tests to determine the average and to decide upon the operation. The cases most suited for tenotomy on the squinting eye are those which average 4 *mm.* My cases averaging 4 *mm* had good results from tenotomy on the squinting eye. Most all those which remained under observation for an average of six weeks got a good position. The minority were divided about evenly into those in which permanent squint was changed into periodic, and complete failure of the operation on the squinting eye, in which case, however, the extent of the deviation had nothing to do with the unfavorable result.

A failure being soon manifest, tenotomy on the fixing eye is indicated. If the operation on the squinting eye has failed, and tenotomy on the fixing eye is done, we can calculate on an effect of 4 to 5 *mm.* The effect may be less, or a permanent squint is converted into a periodic, or in periodic squint the frequency and the amount of deviation is reduced. Or the result may be zero.

Those cases of loss of sight in the squinting eye so that it never fixes are interesting in our study of tenotomy on the fixing eye, and I will here cite some imperfect results of unilateral operations in this class.

Case 15.—Agnes, age six, convergent squint O D for four years. Deviation 6 *mm.* Fingers counted excentrically at 1 *m.* Em O S and perfect vision. Tenotomy of internus and advancement of externus. Ten years later, convergence O D, 4 *mm.* Vision as before, O S M—2 D S. Tenotomy of internus of O S with immediate and permanent good result.

Case 16.—Hermann, age thirteen, convergent squint O S since childhood. Fingers at 1 *m*, O S. Leucoma of cornea O D slight M. Tenotomy internus O S, and advancement of externus O D. Ten days later deviation less. As result was defective, tenotomy on internus O D. Result good.

Case 17.—Miss S., age twenty-four. Convergent squint 8 *mm*, O S for many years. Excentric fixation, fingers counted at 1 *m*. Hm O D + 2. Tenotomy and advancement O S. Sutures tied 6 *mm* behind the tendon, and then ligated to the tendinous insertion. Position good, with occasional convergence. Finally, internus of O D was tenotomized with permanent good result.

I will next cite an example of the different results encountered in this operation. It is also interesting in the point of binocular vision in squint.

Case 18.—Rosa, age fourteen. Convergent squint O D 5 to 10 *mm* ten years. Secondary deviation of O S less, Hm O U. With red glass over O S, no diplopia. Tenotomy and advancement O D. A week later slight convergence. With red glass, homonymous diplopia. Deviation increased in a month to 5 *mm* at 5 *m*.

Patient returned for a new operation three years later. In narcosis slight convergence. Internus O S divided and loosened far back. During narcosis marked divergence. Some weeks later convergence of 3 *mm; crossed diplopia repeatedly verified!* With proper lenses position better, but occasional slight convergence.

The case shows that notwithstanding tenotomy of internus and advancement of externus in squinting eye, and later tenotomy of internus in fixing eye, the result was slight!

TENOTOMY OF BOTH INTERNI.

I have done double tenotomy of the interni in but few cases where the deviation had been measured by prisms. The result was about as in tenotomy on each eye in succession. The result may be slight or zero despite free subconjunctival loosening. If advancement were subsequently done, we find the externus often defective. We can calculate on equalizing a deviation of perhaps 5 *mm.* Here too the direct result does not always correspond with the final, for an originally satisfactory position may retrograde into convergence, or a moderate convergence may decrease, or a slight divergence may disappear. Binocular fixation being lost, it may happen as after advancement, that relative divergence for near, and slight convergence for far, are observed.

The relation of squint to Hm is of special interest. It is well known, *e. g.*, that many cases of periodic squint can be corrected by glasses, whilst others cannot. This is also seen in permanent squint, which is fundamentally the same affection. The deviation in permanent squint being variable, glasses may at times reduce it, and at others be of no effect. So too after an operation. Hence we may make the rule to order glasses before the operation, and watch the effect on the squint. After the operation, also, patients may wear correcting or over-correcting glasses. This procedure is often successful, but not always. The operation may be a success even if we do not correct Hm, until at last by increased tension and alteration in the breadth of accommodation, the condition of affairs is changed. Here is an instructive case.

CASE 19.—A boy of twelve. July, 1873, convergent squint O D. Hm under atropine + 4 D S O U. Double tenotomy of internus gave good result. Sixteen years later periodic squint of 2 *mm* O. D. Hm less than before, but patient had not worn glasses. On giving lenses of + 1.25 D S O U the squint disappeared.

The great variations obtained from simple tenotomy prove that the tenotomy alone is not decisive, but that the strength of the antagonist affects the result. I have said in my text-book that, "after loosening the tendon of a muscle, it retracts as far as its elastic tension demands, and as its connection with Tenon's capsule permits. At the same time the antagonist pulls the eye in an opposite direction till the antagonistic tensions are equalized. But we calculate in tenotomy on sufficient elasticity in the antagonist, and it is plain that tenotomy of the shortened muscle will be of no effect on the position of the eye if there is no antagonistic tension to turn the eye in the opposite direction."

This idea was more fully carried out by Krauchel some years later.[1]

The only means of testing the strength of the externus before the operation is to see how far outward the eye can move. But a lack of such mobility does not mean actual loss of power, as the condition may be due to want of exercise, and can be corrected by the same. *Hence gymnastic exercise of the muscles by alternately turning the eyes to the right and left is a useful preliminary to the operation for squint.* If this fails to increase the mobility, the externus is weak, and this fact is important in deciding whether we shall tenotomize both interni or tenotomize one and advance the externus.

When the deviation is more than 5 or possibly 6 *mm*, double tenotomy is not enough, and advancement is needed. The old way of repeating the tenotomy on the same internus after a bad result has fallen into disrepute. As Liebreich said[2]:

"Whilst the division of the effect into two operations, one on each eye, offered certain advantages for symmetry, and uniformity of motion, the performance of a third or fourth tenotomy, *i. e.*, the repetition of the operation on a muscle already tenotomized, may be accompanied with the greatest inconveniences. Then all calculations cease. The result is in one case zero, in another too great.; so that a well-known surgeon once remarked: 'When we make a third operation we never know how many more we shall have to try on the same patient.'"

With this I agree. If the result of double tenotomy is bad, we do not repeat tenotomy on either eye, but we advance the antagonist.

I will next mention Knapp's plan of increasing the effect by attaching the scleral conjunctiva to the external commissure. This I have tried, but I have failed to obtain any greater effect than by extensive subconjunctival dissection. I have also seen disagreeable redness of the conjunctiva and swelling at the commissure months afterward.[1]

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[1] v. Graefe's *Archiv*, xii., 2, p. 298.

[2] *Ibid.*

TENOTOMY OF THE RECTUS EXTERNUS.

The best cases for testing the result of tenotomy of the externus are those in which binocular vision persists, and in which we can measure with prisms the distance between vertically arranged images and the facultative divergence. The separation of the images is generally less than the facultative divergence, because in the latter the externi are innervated for the sake of binocular vision.

Tenotomy of the externus exhibits as many variations as that of the internus, and the difference between the first and the final result is greater still. The first result may vary from zero to 25°, the final from zero to 9° (angle not prism). The result always decreases. What was at first too great may become zero. I never operate except on cases in which the facultative divergence is greater than the normal. If this rule is not observed and tenotomy is done on the basis of relative divergence, we get an artificial *convergent squint*, as we may see to this day in cases of myopia where an attempt was made to cure the disease by dividing the externus. Illustrative cases:

CASE 20. George, age ten, periodic divergent squint O S since childhood. Normal near point can be reached but with relative divergence. No asthenopia. M 2 D S O U. With lenses and red glass, crossed diplopia at 5 *m*, corrected by prisms 16°. Facultative divergence, 18° prism. Tenotomy externus O S, April, 1878. Good result; gradually decreased later; position good, and no periodic squint. M gradually increased. Eleven years later M — 8 O S. Behind screen, relative divergence or parallelism; normal binocular vision.

Few cases of unilateral tenotomy of the externus remain ong enough under observation to show the final result, and I will cite one or two.[1]

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[1] Sutures passed through the conjunctiva have indeed but little effect, but when passed through the superficial layers of the sclera a more or less marked effect can be produced. I have never seen any unpleasant reaction such as the author mentions, and I shall continue to use the suture as a welcome adjuvant in a certain number of cases.—H. KNAPP.

Case 22. Frank, age sixteen, periodic divergent squint O S; M — 4 D S O U. Facultative divergence 18°. Tenotomy of externus, homonymous diplopia. Later single vision. Facultative divergence 10°. Condition the same a year later. Result of operation 7°.[2]

Case 25. Johann, age sixteen, 1887. Periodic divergent squint O S; Em O U. With red glass, crossed diplopia. Left hyperphoria. Faculative divergence of 30°, at 5 *m.* Convergence up to 7 *cm.* Tenotomy externus O S. Result zero. Facultative divergence as before. Two years later tenotomy of opposite externus. Crossed diplopia fused with prism 4°. Later still, 8°. No deviation behind a screen. The ultimate result was about 15° from two tenotomies.

Those cases in which the deviation can be measured with a prism pass without defined limits into those in which binocular vision is lost, for it often happens that in testing binocular vision the patient's answers are unsatisfactory. The results of simple tenotomy are about the same as in the former class, so that the operation finds the same use as in periodic divergent squint. That is to say, a periodic squint with a deviation of 5 *mm* is relieved, or the recovery is so slight that the other externus has to be divided. Hence where the patients squint nearly all the time but not permanently, and where the deviation measures about 3 *mm*, it is best to tenotomize both externi. Here, too, the immediate result is greater than the final. Slight convergence immediately following the operation generally disappears. When the divergence is great in periodic squint, and the position of the eyes rarely correct, it is better to advance the internus, because the general result of tenotomizing the externus is zero.

INSUFFICIENCY OF THE INTERNI.

In connection with divergent strabismus we have to speak of insufficiency of the interni, but when we employ this term we must first decide what it may include, for Hansen-Grut says that it is an impossible muscular anomaly.[1] If, *e.g.*, the mobility of the eyes is interfered with by their enlargement as in myopia, the defect is more noticeable in convergence than in the lateral movements. Such mechanical obstruction is independent of the muscles. It used to be tested by fixing a distant object with the eyes, then covering one or producing vertical diplopia by prisms, when the covered eye would deviate or we should produce crossed diplopia. This, however, proves nothing in myopia.

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[1] Case 21 omitted for brevity.—Trans.

[2] Cases 23 and 24 omitted.—Trans.

If we so test in Em, the covered eye remains correctly placed, for the accommodative tension which the fixing eye needs to see distinctly at 30 *cm* is divided equally between both eyes, and with it goes a corresponding innervation of convergence. If defective convergence is noticed in Em or Hm with this test, the normal relation between accommodation and convergence is lost, and if the difficulty is removed by prisms we prove that the internus was insufficient for the convergence demanded by near work. This condition, therefore, should be properly styled insufficiency of the interni.

The physiological needs for correct binocular position for near are accommodation and binocular vision. If we make this test in M, at a distance where accommodation is zero, and if we prevent binocular vision by covering one eye or by artificial double images that cannot be fused, it is no wonder that convergent motion is lost. Hansen-Grut does not seem to find these physiological necessities sufficient, and has suggested a "*consciousness of distance*" which compels the covered eye to remain in the correct position of convergence. I really do not know what consciousness of distance is, but why do not most myopes suffer from its loss? If with M of, say, 3 D, we fix an object at 30 *cm*, the covered eye deviates outward, perhaps to parallelism. The exceptions prove that even in M the interni may be stronger than the externi. And at all events, the divergence of the covered eye is largely physiological, and has nothing to do with insufficiency of the interni. When, however, we find that where mechanical obstructions are absent the near point of convergence is reached with effort, and that in the attempt the same eye always lags, we decide that the function of the affected internus is decreased, for the impulse is alike for both.

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[1] *K. m. f. A.*, xxviii., p. 140.

It may also happen that the innervation for convergence is lost, so that the act is no longer accomplished, whilst the associated adduction continues. The affair is still more complicated when divergent squint is also present. When the latter is marked, the movement toward convergence is absent, or is so slight, that the divergence is reduced but a trifle. The proof that innervation for convergence has here been forgotten lies in the fact that in artificial divergence the movement toward convergence is frequently absent. Whenever it is present, but slight, in divergent squint, we find that the adduction of the squinting eye remains too small. We may assume that the innervation for convergence is alike on both sides, as in normal conditions, but that the innervated contraction of the internus finds in the externus of the squinting eye a more powerful antagonist than in that of the fixing eye.

This shows that many conditions are called insufficiency of the internus. Hence the treatment must differ. Where there is no divergence, but defective innervation for adduction, we may use prisms for the relief of the symptoms so far as due to defective convergence. But where divergence exists, an operation is needed. *The relief of insufficiency in divergence by advancing the internus alone*, without tenotomy of the externus, *is generally transitory*. My only success has been:

CASE 26.—Miss K., age eleven. 1877. Divergent squint O S; Hm O U, but V O D fingers at 1 *m*. Convergence up to 20 *cm* only. Advancement of internus. Immediate result, slight convergence. Six months later, position normal. The extreme Hm undoubtedly contributed to the good result.

The result of all the other cases in which I have advanced the internus without tenotomizing the externus has been zero.

ADVANCEMENT.

Tenotomy of the internus proving insufficient for inward deviations over 5 *mm*, and in divergent squint of less, the necessity arose for a more powerful procedure. The need was first perceived soon after the introduction of the operation for squint, for its developmental years were filled with cases of artificial divergence. Jules Guerin first proposed advancement of the retrograded internus, and his case can be found in Desmarres.[1] It was one of artificial divergence for the removal of which the externus had thrice been tenotomized, and probably also resected. Guerin loosened the cicatrix at the side of the externus till the eye could be rolled freely inward with the forceps. Then the cicatrix about the internus was dissected off. In order to maintain the eye in position when rotated inward, a thread was tied close to the cornea, drawn inward, and fastened to the nose with plasters. v. Graefe[2] modified the operation, and used it largely under the name of the thread operation, in artificial and paralytic squint.

Critchett's method of curing artificial divergence is thus done[3]: The cicatrix over the internus is completely dissected away, the stump of the muscle brought forward, a small portion of conjunctiva cut off, and after tenotomy of the externus the internus is tied to the conjunctiva remaining at the margin of the cornea. v. Graefe in 1863[4] extended the indications of advancement in divergence. Liebreich later recommended[5] a broad vertical incision in the conjunctiva near the insertion, then the conjunctiva was loosened on the one side to the periphery, and on the other to the corneal margin. After detaching the muscle, two threads were passed through the conjunctiva at the corneal margin, and then into the entire border of muscle and capsule combined.

It is extremely necessary, as I first asserted,[6] to *secure the muscle before we loosen its tendon from the sclera; for if we tenotomize before securing it, as used to be done, it withdraws of its own elasticity, and we have to fish it out fibre by fibre with forceps, in order to apply the sutures properly.*

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[1] *Maladies des Yeux*, 1858, tome iii., p. 684.
[2] v. Graefe's *Archiv*, Band iii., p. 340.
[3] *Lancet*, May, 1875.
[4] v. Graefe's *Archiv*, ix., p. 48.
[5] Ophth. Congress zu Heidelberg, 1868.
[6] K. Gess. d. Wiss. zu Goettingen, 1870.

The method that I now employ is this: I first tenotomize the squinting muscle, then grasp the conjunctiva over the insertion of the antagonist, and make an incision parallel to the same. I divide Tenon's capsule similarly. The muscle is next raised with a flat blunt hook, which is pushed through the capsule, and everything above the hook is dissected off till the tendon comes into plain sight, resting on the hook. In order to be sure that we have the entire tendon, a second hook is passed under the first, to see if perchance the latter has not pierced the tendon without taking it up *en masse.* The sutures are not to be inserted till we can see the tendon in its full extent. One hook is now pushed toward the margin of the cornea, till it lies beneath the tendon, and rolls the eye in a direction antagonistic to the muscle, and the second hook is pulled toward the periphery till it passes the spot where the sutures are to be inserted. One needle is now passed under the upper margin of the muscle, and then pushed through below the middle from behind forward. The second needle is passed from the lower margin, and pushed through above the middle of the muscle. In this way the muscle is ligated. Each suture has two needles. In this way the muscle lying between the place of ligation and the insertion is eliminated, and I have long entitled advancement as a means of shortening the antagonist, and so of increasing its tension. The amount of shortening to be given to the muscle is measured by the distance at which we insert the threads behind its insertion. This is best done with a small millimetre scale like a strabometer, the zero of which is placed on the tendinous insertion, and the millimetric portion on the muscle. If we shorten more than 5 *mm*, we must dissect the conjunctiva from the muscle toward the periphery. All measurements should be made without stretching, as the ligated portion may turn out shorter than we think. After tying both sutures, the portion ligated should be measured once again without tension, and then the tendon divided. I used to fasten the muscle close to the cornea, but not simply to the conjunctiva, for I long ago insisted that the sclera should be exposed by dissecting off the conjunctiva so as to make a proper surface to which the tendon could adhere. More than that, the conjunctiva is often so defective that it gives no support to the stitches. Therefore I suture the muscle to the *tendinous insertion*, whenever the strength of the conjunctiva seems doubtful. In doing this, we have to divide the muscle near the point of ligation, in order to obtain a portion of the insertion sufficiently broad for attaching the sutures. I have employed this method more and more, till now I universally tie the muscle to the insertion, because to carry the threads to the corneal margin produces unwelcome distortion of the conjunctiva. This is to be feared if we excise some conjunctiva, but with the latter we must be sparing, as it cannot easily be restored.

We can remove a piece of the muscle if ligated far back, as there is no need of leaving more of the muscle on the insertion than is required for suturing. But generally there is no need of any excision, and the amount removed is of no importance so far as the result is concerned, for the amount of shortening is actually determined by the distance at which ligation is done behind the insertion. After dividing the muscle the fibres retract toward the insertion, and since for the very purpose of tying them to the tendon the threads are passed close to the sclera through the tendinous ridge, the aim of shortening the antagonist is attained without excising any of the muscle. The result obtained by advancement in convergent squint is about the same as the shortening of the muscle by the operation. I therefore ligate from the muscle an amount equal to the average linear deviation. If the latter has been correctly measured an over-effect is rare, yet we may see the same variations as after tenotomy. If the result is too small we can resort to tenotomy or advancement on the fixing eye. It makes no essential difference in the operation, whether we attach the muscle to the subconjunctival tissue or to the tendinous insertion, but the latter gives the most accurate basis of measurement.

I have never advanced Tenon's capsule, though it cannot be denied that with this operation we can exercise indirect traction on the muscle. Still it is not a step that can be measured, as in direct operation on the muscle itself.

The only cases fit for comparison are those which remain under observation for at least a month. Comparison thus made after advancement, in which the distance of the sutures was measured from the tendon, and the muscle brought forward to the old insertion (with tenotomy of the squinting muscle), showed that *the average result was about the same as in shortening the antagonist.* When the result was less, the antagonist was weakly developed. One case of too great effect was not seen again.

We also observed that after advancement a permanent convergent or divergent squint may be changed into a periodic.

Advancement may be done by passing a double-threaded needle through the muscle after division, and ligating the sutures above and below the cornea till the eye stands correctly. The widespread view that any advancement can at once put the eye in a position which it shall permanently maintain rests on false premises. Just as in tenotomy so in advancement, the immediate result does not always remain permanent. The original result generally diminishes, yet here is an exception.

CASE 31.[1]—Alternate convergent squint, 5 to 7 *mm*, with slight upward deviation O D, and slightly downward O S; Hm O U. Has worn proper glasses two years. October, 1893, double tenotomy of interni, with subconjunctival loosening. Result poor. Then tenotomy of internus with advancement of externus O S. Abducens weak. Ligation 5 *mm*. Two months later, position bad. In May, 1894, position good, with slight periodical convergence.

The same rules affect advancement of the internus in divergent squint. The result is often less, and we must tenotomize the externus, and advance the internus of the other eye. When the deviation was more than 5 *mm* I used to combine double tenotomy of the externi with advancement of the internus in the squinting eye, and with good results, but this I have abandoned the more the technique of advancement has been perfected by exact measurements and suturing to the insertion. If the deviation averages more than 5 *mm*, we must from the first keep in mind the probability of an operation on the fixing eye, but we cannot know beforehand whether tenotomy alone will suffice or the internus also must be advanced. For the result of the advancement, even if the internus is shortened 5 or 6 *mm*, may be poor. This is known soon after the operation, and if visible four weeks later will in my opinion remain permanently so.

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[1] Cases 27 to 30 have been omitted for brevity.—TRANS.

Advancement helps us to discover a weakly developed antagonist, as I first suggested,[1] and as Schneller confirmed,[2] asserting, in addition, that the squinting muscle is simultaneously over-developed. This, I have no reason to doubt, but I cannot confirm, as I never operate so as to see the muscle. I simply see the whole of the tendon. But I am well aware that the tendon is often more developed in the squinting muscle than in its antagonist. Weakness of the latter cannot be due to the squint, because with marked deviation we often see a normal antagonist. More than this, the fact that tenotomy of the squinting muscle may have no effect even in slight deviations proves that we have a right to add weakness of the antagonist to the etiology of squint.

Both eyes may have weak muscles, which would explain why double tenotomy or tenotomy on the fixing eye is often fruitless. These relations are also complicated by differences in the refraction or vision of the eyes. Congenital or early acquired weakness of an ocular muscle may exist in the eye with the better sight, which will then be the fixing eye. In order then to maintain correct fixation the weaker muscle will need stronger innervation, which by association will be transferred to the other eye and cause it to deviate. As proof of this I add a *peculiar case of artificial squint on the eye that had not been operated upon.*

CASE 32.—Miss H., age twenty-four, May, 1894. Artificial divergent squint of the RIGHT eye. Deviation 5 *mm.* Fingers at 1 *m.* Convergence up to 15 *cm*; Hm : O S + 2.5 D. *Tenotomy of internus O S had been done some years before, leaving artificial*

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[1] *Untersuch. u. d. Schielen*, 1881, p. 128.

[2] v. Graefe's *Archiv*, xxvi., p. 3.

divergence in the other eye. The disfigurement was removed by tenotomizing the externus and advancing internus. The cicatrix of the old operation was plainly visible.[1]

I was puzzled to understand why tenotomy had ever been done on the left eye. Here we see experimentally that the weakness of the internus, owing to the stronger innervation needed for fixation, was followed by associated divergence of the imperfect right eye.

ARTIFICIAL DIVERGENT STRABISMUS

may originate in many ways. A sunken caruncle, reduced adduction, and difficulty in isolating the internus demonstrate a defect in the previous tenotomy. The same condition may arise if with a perfect operation the measurements were incorrect, and especially in convergent periodic squint, because the deviation varies so much at times. Another cause lies in the variations appertaining to all methods of operating. Finally as Weiss has shown,[2] the alterations in the skull by age, play an important part. The latter, too, may explain the spontaneous cure of squint as well as those rare transformations of convergence into occasional divergence. But just as a convergence may spontaneously disappear, so can a good result become artificially divergent. Many cases of convergence show slight divergence soon after the operation, but it vanishes or leaves a slight convergence. So a slight divergence may remain, but it is harder to see than the apparent divergence in Hm. Generally, however, we call "*artificial,*" only those cases with distinct divergence, and loss of motion toward convergence. And here I cite:

Case 33.—Max, May, 1888. Convergent squint 8 *mm* O S; Hm O U. Tenotomy and advancement O S. Result 4 *mm* convergence. Later, tenotomy internus O D. For two years position was good, but then divergent squint O. S. Tenotomy, externus, advancement of internus, O S. Result, good.

Case 34.—Gertrude, age twelve, periodic convergence O D 5 years, uses + 2 D S O U, but lately squint has become permanent. Both interni tenotomized October, 1890. January, 1891,

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[1] How about the weakness of the left internus?—H. K.

[2] Ophthal. Gess. zu Heidelberg, 1893.

position good, minimal divergence. In 1893, Hm had disappeared and Em present: visible divergence O U. No convergence! The innervation of convergence had been forgotten. Divergence being marked, convergent impulse absent and vision perfect without lenses; in 1893 tenotomy of externus and advancement of internus were done with good results.

The changed shape of the skull may have been active in these cases, but without this influence surprising changes are seen. Thus:

Case 35.—Bertha, age twenty, has had convergent squint 19 years in O S; Hm. Tenotomy of internus and suture at external canthus. Convergence less. Later, tenotomy of fixing eye. Less convergence. Repetition of tenotomy O S. Later, divergence. Six months later, tenotomy of externus O S. Position, good. Crossed diplopia with red glass and vertical tests.

It would have been better to complete the defective result in the squinting eye by advancing the internus, but as the result of tenotomy on both eyes was slight, the subsequent divergence is surprising.

UPWARD AND DOWNWARD SQUINT.

Vertical deviation occurs most often with lateral, and disappears after the removal of the latter. Hence it is not surprising that after the operation for convergent squint, especially, we see a vertical deviation which before was not observed or did not exist (Case 3).

The frequency of this occurrence has originated the rule that with vertical and lateral deviation, the latter should first be attacked. If the former remains, it may be operated separately. Two muscles, a rectus and an obliquus, being concerned in vertical deviation, the former alone being accessible to operation, if we tenotomize either the superior or the inferior rectus, two antagonists have an influence on the result, so that uncertainty ensues. The average results are good. An over-correction is rare.

In most cases the squinting eye deviates upward, but I have seen one of deviation downward.

Case 36.—Gustav, age twenty-one, 1891. Divergent squint O D 5 *mm* with deviation downward. No convergence, M O U. Tenotomy of externus and advancement of internus O D. Result good. No diplopia with red glass or vertical prisms.

Simple vertical deviation without lateral is so rare that I will append a case.

Case 37.—Elizabeth, æt. twenty-three. Upward squint of 4 *mm* O D, since childhood. Secondary deviation downward of O S. April, 1889, tenotomy of superior rectus O D and advancement of inferior, 5 *mm* being ligated. The inferior was feebly developed. Result good. Screen-deviation out and up; in other eye out and down. No diplopia with red glass.

We must not forget that in vertical deviations we meet with obstacles to the laws of innervation. If an eye deviated upward fixes, it moves down, and the other ought to make a uniform movement. But there are exceptions. One eye may move up and in, and so become the fixing eye, whilst the other shows associated adduction without vertical motion. Or with deviation up, the secondary deviation of the other eye may be upward also. For example:

Case 38.—Hedwig, age nine. Alternating convergent squint with upward deviation. The former slight, the latter 4 *mm* and the secondary deviation of the non-fixing eye is also up; Em O U. Double tenotomy of interni. A few days later the convergence had disappeared, but the vertical deviation persisted. Six years later, same state.

Bilateral deviation upward, being contrary to the laws of innervation, we can only assume elastic forces stronger than any innervation.

BINOCULAR VISION IN STRABISMUS.

In the best sense of the word, squint is only CURED with the restoration of normal conditions, including binocular vision. Still, the operation can only do what is possible. Whether any use will be made of binocular vision depends on its condition before the operation. It has long been known that diplopia was rare in squint, the fact being explained by suppression of the retinal image. More properly speaking, failure to observe the retinal image is common enough. Many a friend passes us in the street without our observing his retinal image, but we have no reason to complain of suppression.

Squinters often know that they fix with one eye and neglect the image of the other. But it is easy to prove that this is not habitual as the theory demands. We have only to cover the fixing eye with a red glass, and the other with a vertical prism, to produce in most cases a diplopia, with less lateral deviation, however, than in normal binocular vision.

If this test fails we can try mine[1] in which the fixing eye is directed at a distant flame. Lying to one side and behind the squinting eye is a second flame, the image of which is thrown into the eye by a plane glass held close to the latter. We see this reflex on the cornea, and by tilting the glass so that the reflex is brought into the centre of the pupil, the patient sees it also and can describe its relation to the other image.

A third test is to cocainize the squinting eye and to place it in a position opposite to its usual direction with forceps, when diplopia will be produced.

In a word, there are many ways of showing that there is no permanent habitual suppression of the retinal images in the squinting eye. When red glass fails, we can discover diplopia by vertical prisms. Or, with prisms of varying strength, we can determine where, in the retina of the squinting eye, diplopia is not perceived. This is v. Graefe's "*regional exclusion*," which I do not deny, but which might as well be called regional single vision, instances of which have been published. The development in long-standing cases of squint of peculiar relations between the retinæ, relations lying outside the laws of normal binocular vision, is proved by the frequency after an operation of diplopia which was not present before, and by the fact that the images are alleged to *be crossed when the eyes are actually turned in!* This may disappear, but there are cases in which it is a disturbing element for years, despite the use of prisms. Oftener, of course, diplopia is not complained of, but it can be produced by red glass, though not always corresponding to the laws of identity. *E. g.*, a patient with divergent squint will see homonymous images. This may not be precisely diplopia, but it is alternate vision, in which the image seen with the left eye is asserted to be at the left, and *vice versa.* If crossed diplopia is alleged with eyes turned in, the fault may be due to the fact that the squint has produced relations of the eyes to one another which when they were squinting maintained single vision, but when the old position is altered by the operation, diplopia ensued. Moreover, right and left being confounded by surgeons, why should not the patients make the same mistake?

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[1] *K. m. f. A.*, 1876, 22.

Similar false or inexplicable answers are heard in vertical deviation. Thus:

Case 39.—Miss W., who had been operated upon six years before by double tenotomy of the interni, consulted me in 1891 for frequent upward deviation of O D. Under cover, divergence and deviation upward of each eye, but less O S than O D. With red glass, crossed diplopia, right image higher. Prism 9° base up before O D levels the images. Operation declined. Next year same condition, but O D occasionally deviates up. When covered, deviation further upward, and the eye remains fixed so. O S, covered, deviates up, but returns. With lenses for Hm and red glass, image of O D higher. Prism 7° base up O D levels images. Stronger prisms lower image of O S. According to laws of identity, image of O D should have stood lower. Tenotomy of superior rectus O D had good result for the few days that the patient remained under observation.

Binocular vision in squint is perfectly explained from an empirical point of view. Binocular fixation has an anatomical foundation, for the fovea centralis is the best portion of the retina, and hence should be used for fixation. If this has once become a habit, retinal identity develops of itself. But if binocular vision is acquired, it can be forgotten, especially when it has only for a short time been exercised.

Case 40.—Marie, age six, had marginal keratitis O S in 1878; atropine treatment. Blepharitis ensued; atropine stopped. In three weeks convergent squint developed in O S and became permanent. Hm O U. Homonymous diplopia was complained of without suggestion. One month later squint the same, but no diplopia. + 2 D S O U. Six weeks later no convergence. Red glass, homonymous diplopia. The squint soon disappeared, and has never returned. Glasses were worn for a while and then laid aside.

It is hard to determine the cause of this condition. The Hm could not have been due to atropine, for the child had already learned to read without squinting, and was not using the eyes for near work when the squint appeared. Nor can we see any reason in the keratitis; for this was better before the squint began.

Still another contradictory condition of binocular vision is seen in :

Case 41.—Bertha, aged eighteen. Asthenopia. Em O U, and perfect vision. Under cover, divergence. Patient remarked that people said that she occasionally squinted. Normal binocular fixation. On covering either eye, it deviated out. Convergence normal to 9 *cm*, where O D in binocular fixation lingers. Hence, though binocular fixation was proved, binocular vision was absent. For, with red glass or vertical prism, diplopia was observed with visible divergence of the eyes. On fixation at 30 *cm*, homonymous diplopia was alleged, though the eyes were divergent. This showed alternate, but not binocular vision. The stereoscope showed that the left field only of the squinting eye was seen. Yet Hering's test was positive.

The asthenopia was due to conjunctivitis. Tenotomy of right externus had but little result.

In periodic squint the opposite occurs : the images are correctly placed, but the stereoscope shows that only the field of the fixing eye is seen. Hering's test is negative.

If squint originates at an age when binocular vision has become habitual, we can generally demonstrate double images which agree with the laws of identity, and the operation gives double fusion. This may also occur where double images could not by any means be proved before the operation, a point which shows that patients' assertions must be regarded with distrust. Thus in fresh ocular paresis the patients at times cannot be made to see double. Or in the relative divergence of M, diplopia is observed for far, but not for near, which proves defective observation of the people with whom we have to do, and warns us not to build theories on assertions that may not be correct.

Squint originates mostly at an age when binocular vision has not become habitual, and even if the operation gives a position which renders fusion possible, it is not acquired.

The solution is simple: normal binocular vision is physiologically correct, but squinters are used to a sort of binocular vision which they have acquired, and in this the disadvantages of one-eyed vision are not perceived, presuming, of course, that the squinting eye has some vision.

One of the results of the suppression theory was the loss of sight from non-use. Suppression destroyed the perceptivity of the eye. But when we show that there is no permanent suppression, the theory falls to the ground. Nor does it agree with the state of affairs, for loss of sight beyond $\frac{1}{7}$ is only seen in a third of the cases. Higher degrees are rare, and may be seen in eyes that do not squint. Cases, too, in which the defective eye with inward deviating visual axis is the fixing eye, occur in convergent or divergent squint, or even where there is no squint at all. Yet, again, if the loss of sight in the squinting eye were a sequence of squint, it ought to be progressive. It should increase with the duration of the squint, which again is not the case. More than that, it should increase even after the operation, for the latter does not often produce binocular vision.

Cases of improved sight after the operation should be taken circumspectly, for squinters who have never learned to use the weaker eye for fixation often answer incorrectly. Only repeated and harmonizing tests for far and near have any value. Personally, I have never seen any improvement in vision in a squinting eye after an operation, or even after separate exercising.

Circumspection also is the rule where eyes with previously good sight have been injured by the squint itself. Almost all of these occur in children, who at the first tests had good vision, but in after years asserted that the sight of the squinting eye had grown worse. But such a lamentable accident I have often enough *seen occur in a few minutes.* Every test case has a disc to cover the eye that is not under examination. But on this disc we cannot rely. I have seen adults, who knew nothing of their congenital loss of sight, who with the disc before the normal eye had better vision than when the eye was actually closed. Still more is this so with children. It is useless to cover the fixing eye with the hand, for they peep through the fingers. Even a handkerchief which is offered to them rolled up is held in such a way that they can look alongside the nose with the good eye. The only way to prevent fraud is to let a third person press a handkerchief well against the nose so that no sight can enter there. All visual tests are subjective answers of people whose capacity for observation is small, and before we use these scientifically, we should employ every possible means to prevent unconscious falsehood.

STRABISMUS THEORIES.

Theorists would like to explain everything from one point of view, but all things that happen, happen from many causes, and this is true of squint. Donders's idea, that most cases of convergence occurred in Hm, and most of divergence in M, was a valuable enrichment to science, but when we conclude that the connection has no other basis than through the accommodation, we are going too far. For hypermetropes learn to accommodate with less innervation of the interni than emmetropes. The plain fact that most hypermetropes do not squint led Donders to think that this depended on the weakness of one eye. Now my investigations show that in Hm where one eye is weaker than the other only 53 per cent. squint, whilst as many as 47 per cent. do not, and that in spite of the most favorable opportunities to do so. Therefore there must be other causes for squint, and the next place to look for them is in the muscles. Still we must not neglect the fact that tension of the accommodation plays a great rôle, for there are cases of periodic squint in which the eyes are straight on wearing glasses. And even permanent squint may disappear in the same way. Therefore lenses should be tried as well before, as after, the operation.

Donders states that hypermetropes can choose between binocular vision with full use of accommodation and the asthenopia which that causes, or monocular vision with squint and an accommodation corresponding to the greater convergence. But convergent squint generally appears in childhood, and even high degrees of Hm do not disturb little children. It is only when they begin to go to school that we are asked about their "shortsightedness," which turns out to be Hm.

The connection between Hm and convergence, and M and divergence, cannot be denied, but accommodation is rarely the cause. Even when there is no squint in M, the facultative divergence is greater than in Em, probably from stronger externi, which when developed in higher degrees may excite divergent squint. M and Hm being opposite conditions we may draw similar conclusions concerning the interni in the latter, though the convergence commonly present when testing the equilibrium in Hm may be due to accommodative tension. The accommodative theory having become untenable in most cases we had to look about for another explanation, and Stilling came forward[1] with the assertion that the so-called position of rest was the position of the squinting eye and his investigations showed that in most cases of Hm, convergence, and in M, divergence, was the position of rest.

According to Hansen-Grut[2] convergent squint is "Innervation," and divergence is "Position of rest." The latter we find by closing one eye and simultaneously fixing a distant point with the other or by aid of a vertical prism. Even then there is doubt, as the closed eye may remain parallel to the other and yet have the anatomical position of rest which is due to "Habit."

"Habit" is obtained by binocular vision, but if this is prevented by vertical prisms we should not expect it to influence the position of rest, for no one has ever habituated himself to tests with vertical prisms. Still, in interest of the theory, the normal condition must be overcome. If the tests show parallelism, this cannot be the normal expression of muscular tension, but a "functional" position of rest, due to unconscious habitual tension of the interni to overcome a secret divergence, for the latter must be the anatomical position of rest, otherwise the theory would be of no use for divergent squint. We are not told how this position is gained in each case. The author simply suggests that after death, or in narcosis, the eyes diverge. The eyes can be easily seen in narcosis, and are much distorted. Whether this corresponds to the anatomical position of rest I do not know, but I do know that neither death nor narcosis is the natural condition of man.

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[1] These ARCHIVES, xv., 270.

[2] See present number of these ARCHIVES for Hansen-Grut's theories in abridged form. Or *K. m. f. A.*, xxviii., p. 134, for the same in full.

"Innervation" of the interni so necessary to the position of rest is aptly compared with latent Hm, which once meant permanent tension of the accommodative muscle (improperly, spasm of accommodation), but objective determination of the refraction proves that there is no permanent tension of this sort. When this can be done without mydriasis, we find the actual Hm, only the other eye must be closed. Hence latent Hm is absent when hypermetropes have no chance for fixation. It is only when they want to see clearly that they stretch their accommodation, and often cling to the habit even when glasses are worn.

Permanent convergent squint is, in Grut's opinion, maintained by "unconscious innervation" of the interni. Occasional Em in convergent squint is explained by the infantile Hm being easily overcome by accommodation. If so, we should think that the innervation of the interni would persist even when Hm had long since been transformed into Em, and average accommodation alone remained. If so, then almost every one would squint, for Hm is the most frequent refraction in childhood.

Hansen-Grut says "innervation," and protests against anatomical shortening of the squinting muscles. Nevertheless, all muscles act so, becoming shorter when at rest, and if the squinting muscle maintains a permanent deviation it must be anatomically shorter than in its normal condition.

Now there is no such "permanent innervation" as this. For every muscle (except the heart and the respiratory muscles) which is permanently innervated grows tired and painful. This may not happen in squint, but what shall we say of asthenopia as it occurs in all errors of refraction, as shown by the relief obtained by prisms? So, too, occasional pathological innervation of single muscles, as in facial spasm, differs *in toto* from what we have in periodical squint.

Hansen-Grut supports his "innervation" by a "tonus"; but what he calls "tonus" is the physiological state of rest of normal muscles in contradistinction to paralysis or death. This is well shown in toxic paralysis of the ciliary muscle by atropine, which offers at the same time a chance to measure the difference between physiological rest and paralysis. It generally amounts to 1 dioptry.

Further still, divergent squint is asserted to be the anatomical position of rest, but we have just seen a case of artificial divergence, which could not be due to anything else than an active associated contraction of the externus. This must be so when the fixing eye with better vision also has an imperfect internus.

The additional assertion that binocular vision must be abandoned in absolute divergent squint is wrong, for we see many such patients enjoying binocular vision.

Then again, the fact that tenotomy of the externus in absolute divergence has less effect than that of the internus is no proof of the innervation theory, for the internus is well innervated in such a case. The associated innervation is not affected; it is simply the innervation of convergence that is weakened.

We must next consider that, according to Hansen-Grut, all blind eyes ought to remain in the anatomical position of rest. Unilateral or bilateral blindness in cataract is not rare, but is divergence the most frequent position of the eyes in such cases? (Yes.—H. K.)

All muscles, whilst maintaining their function, are innervated, and they also have an elastic tension. Then the question arises how far these two points are affected in squint. *The influence of accommodative innervation is also felt in permanent convergent squint by increased deviation when the accommodation is tense.* This is, however, chiefly shown in periodic squint, of which I have seen many cases with Hm in which the finest type could be read without glasses, and even near to without a squint. Still, in periodic squint, convergent as well as divergent, innervation plays an important rôle, passive rather than active. Thus: periodic squint occurs when people are tired, and when the innervation is reduced so that the elastic forces gain the upper hand. This is especially seen in narcosis, in divergence of eyes that never squint, and in a convergent squint disappearing. If, as also happens, convergence persists under narcosis, it cannot be due to paralyzed innervation. Hence there must be elastic forces at work (to be first sought for in the muscles, and even outside them, as in upward deviation of both eyes), which exercise some influence in the production of squint.

Those who pay attention to advancement operations know that the antagonist of the squinting muscle is often poorly developed. High degrees of this we can see with the naked eye, and it is much to be desired that Mueller's observations[1] in this province of squint might be verified by microscopic investigations. Schneller, too, has seen enormous development of the squinting muscle. And so when Hansen-Grut says "that the talk of weak and strong muscles in squint is a mere collection of words without a shadow of foundation," it seems to me that this bitter phrase might much rather be referred to his theory of squint.

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[1] *K. m. f. A.*, April, 1893.

TUBERCULOUS INFECTION OF THE EYE.

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Translated by Dr. Robert L. Randolph, of Baltimore, Md.

(*With Plate II. of Vol. XXVIII., German Edition.*)

FOR years Michel has emphasized the fact that tuberculosis exists much more frequently than is usually supposed as an etiological factor in the various eye affections, and especially in the diseases of the uveal tract. In opposition to other text-books on ophthalmology, in which tuberculosis of the eye is treated of as a rare affection, it will be found that in the second edition of his text-book, Michel has not only given this question a position of importance, but has actually proved it to be one of such. This opinion, too, has found expression more than once in the various dissertations that were prepared under his direction. It is possible, indeed very probable, that in lower Franconia, where tuberculosis is rife, one has a better opportunity of studying the tuberculous affections of the eye, and hence my reasons for bringing forward these observations.

Before I describe the way in which tuberculosis shows itself in the different coats of the eye and its appendages, I shall lay stress upon the following four points.

1. Tuberculosis of the eye is by no means a rare affection.

2. All parts of the eye may be attacked by the disease.

3. It plays a particularly important rôle in diseases of the uveal tract.

4. The eye disease may be the only and earliest manifestation of the tuberculous infection.

I have seen tuberculosis of the *lids* in three different forms, and the clinical diagnosis in many cases has been verified by the transplantation of some part of the diseased lid into the anterior chamber of a rabbit. I have occasionally observed lupus of the lid occurring either as an isolated affection, or having extended to that part from similar disease of the skin of the nose or cheeks. There are very rare cases too in which the so-called chalazion consists of tuberculous granulation tissue, and the tissues may be attacked by tuberculosis in a more diffuse manner still. Of the truth of the last statement the following case is a proof.

A lad, seven years old, exhibited an almost complete ptosis on the right side. The upper lid was swollen, and had an extremely soft, spongy consistency to the touch. It was only over a small area at the medial end that the tarsus showed any of its normal character on palpation. There were no appearances of inflammation on the skin of the upper lid. The tarsal conjunctiva, as well as the retrotarsal fold was succulent and swollen, resembling granulation tissue. There was a moderate mucous secretion from the conjunctiva.

The drooping of the lid progressed very slowly. The diseased tarsal tissue was excised, and a piece was examined microscopically. A piece was transplanted into the anterior chamber of a rabbit, and in four weeks an extremely well-marked tuberculosis was present, and large numbers of tubercle bacilli were found under the microscope.

In the *conjunctiva* the clinical and anatomical picture of tuberculosis may vary. Sometimes we have the tuberculous ulcer in different parts of the conjunctiva; then, again, especially in the tarsal conjunctiva, we find the yellowish-red miliary nodule. This latter form may be either of short duration, or it may lead to the formation of a tuberculous ulcer. Lastly, we may find, particularly in the fold of transition, but also occasionally in the scleral conjunctiva, follicles infected with tuberculosis. The form just described may simulate perfectly the anatomical and clinical picture presented by trachoma.

A girl, fourteen years old, came with the complaint that her left eye had been troubling her for the past few weeks. The condition was as follows : The left lower lid, chiefly on the nasal side, thickened and showing inflammatory infiltration ; slight eversion of the border of the lid. In the tarsal conjunctiva, at a short distance from the border of the lid, there was an ulcer with elevated, irregular borders and a lardaceous coating. Granulation tissue : a piece was excised, and transplantation gave a positive result. The ulcer was made to cicatrize through cauterization.

Neither the examination nor history gave any ground for the diagnosis of syphilis, and potassium iodide proved useless.

The following may be regarded as a classical case of tuberculous disease of the eye and its appendages.

A carpenter, thirty-nine years old, came with the history of having had an inflammation of his left eye for fourteen days. The secretion of tears, which in its commencement had been associated with slight pain, was increased, and the eyelids were stuck together in the morning.

R. Anophthalmus.

L. Conjunctiva succulent throughout ; moderate muco-purulent secretion ; in the temporal portion of the tarsal conjunctiva of the upper lid, as well as in the fold of transition, there were numerous miliary nodules thickly crowded together, and of a grayish-yellow appearance. On its lower border the cornea showed a semilunar, deeply situated opacity, over which were minute areas in which the epithelium was absent (maceration), alternating with spots in which there were proliferation of epithelium and, in places, a new formation of vessels. Near the lower and outer border of the cornea there was a little nodule in the sclera tolerably prominent, and of a yellowish-red color. In the vicinity of this nodule there was circumscribed scleral injection. No nodules visible in the ligamentum pectinatum or iris. There were numerous posterior synechiæ. In the pupillary area there was a delicate gray membrane. The fundus reflex was much diminished, and the details could not be seen. V = fingers at $3\frac{1}{2}$ *mm.* The nasal region on the left-hand side and the neighboring parts of the cheek were moderately swollen, there being probably tuberculous periostitis in this region.

General condition : Catarrh of the apex.

A piece of the diseased tarsal conjunctiva was excised, and transplantation gave a positive result. Bacteriological examination showed no tubercle bacilli. The tuberculous nature of the disease, however, was proved by the result of the transplantation. The little nodule in the sclera had certainly a like origin, and the corneal clouding can be attributed with a probability bordering upon certainty to primary tuberculosis of the ligamentum pectinatum, with secondary involvement of the cornea. The old iritis was also tuberculous in its nature. Any doubt in the matter is dissipated by the pathologico-anatomical condition of the right eye. A detailed description of the latter with an illustration may be found in the inaugural thesis by Emil Bongartz in 1891. The eye was enucleated by Professor Michel, on account of the excessive pain in it.

The clinical condition then was as follows: R. Vision = ability to see movements of the hand when held as close as possible to the face; marked circumcorneal injection; diffuse parenchymatous clouding of the cornea, and the latter was the seat of numerous blood-vessels; the anterior chamber was shallow, and the iris discolored. The pupil was closed by a connective-tissue membrane. There were both exclusion and occlusion of the pupil. The intra-ocular tension was diminished. The slightest pressure on the eyeball produced great pain.

Pathologico-anatomical condition: The epithelial layer of the cornea was unchanged. At the corneo-scleral junction, and right under the epithelium there were to be seen occasional masses of leucocytes. The lamina elastica anterior was in a good state of preservation. The parenchyma of the cornea was infiltrated with great numbers of leucocytes. In the middle of the thickest part of the parenchyma of the cornea there were numerous new-formed blood-vessels, surrounded by ensheathing spaces filled with masses of leucocytes. The vessels were most numerous on the border of the cornea, and grew fewer in number towards the centre. Both Descemet's membrane and the endothelium were in a good state of preservation. At a point corresponding to the space of Fontana, and from this point towards the middle, the posterior surface of Descemet's membrane was devoid of its endothelium, and was bound down to the upper surface of the iris by pretty broad bundles of fibrillated tissue (see Plate II. *B.*). In this tissue there were little spaces lined with endothelial cells, and containing great numbers of leucocytes. At other points the posterior surface of Descemet's membrane was adherent almost directly to the surface of the iris (see Plate II. *D* in the upper, and D[1] in the lower half), and at the points of adhesion there was a delicate fibrous tissue slightly infiltrated with leucocytes. Just here the membrane of Descemet was thrown into irregular folds. Within the network of the space of Fontana there were large numbers of leucocytes. The anterior portion of the sclera showed infiltration with leucocytes. Small hemorrhages were to be seen along the course of the larger vessels. As a rule the open vascular spaces contained an excess of leucocytes. At some points there were circumscribed masses of epithelioid cells and leucocytes.

The iris appeared widened, this being due to an abundant infiltration with lymphoid elements and proliferated flat cells. The pigment layer in some points showed increased growth, and in other places was atrophic. Circumscribed accumulations of cellular elements, which could be regarded as tuberculous nodules, were extremely rare. They were found in greater numbers in the ciliary body, in some of the ciliary processes, particularly between the bundles of muscular fibres at the points of attachment in the latter, and in the sclera in the immediate neighborhood of the ciliary muscle (compare Plate II). These nodules were most numerous in the orbiculus ciliaris and on the adjacent choroid (compare Plate II), and at these points they appeared under the form of confluent tubercles. At this point the suprachoroidea was free, but, on the other hand, the inner layers of the sclera in the same situation were beset with tuberculous nodules.

In positions corresponding to the points just mentioned, the adjacent retina showed a high grade of connective-tissue growth in which were to be seen tuberculous nodules (compare Plate II). Farther back from this point a direct extension of a nodule of the choroid into the retina had taken place (Plate II). The pigment-epithelium is in this situation either displaced laterally or is still visible within the nodule. The remaining portion of the choroid was likewise infiltrated with numerous nodules. Sometimes the nodules would take in the whole depth of the choroid and cause a thickening of the latter to three or four times its original size. The smaller nodules had the shape of a cone with its apex directed toward the retina. In some places the nodule had originated in the choroid proper, the suprachoroidea remaining free, while in other places the suprachoroidea alone was involved. The adventitia of the numerous larger blood-vessels was quite often the point of origin of the smaller nodules. As already remarked, tuberculous nodules had broken through the choroidin to the retina, and after destroying the neuro-epithelial layer had come into direct contact with the other layers of the retina. In other places tuberculous nodules, which had no connection with tubercles of the choroid, were present in the internal granule layer, nerve-fibre layer, and ganglion-cell layer (Plate II). Sometimes these nodules would take in scarcely more than the thickness of the internal granule layer; in other cases would involve the entire thickness of the retina, or even cause considerable increase in the width of the latter. In some places there were to be seen nodules at corresponding points in the choroid and retina without there being any direct connection between the two. The optic nerve as far as could be seen was the seat of tuberculous disease (Plate II). A tuberculous nodule was found just at the posterior limit of the lamina cribrosa, and lying next to the central canal, and at several other points in the nerve tuberculous nodules were present.

The microscopic examination of these nodules showed that they consisted of epithelioid cells, giant-cells, and leucocytes. In the retina and optic nerve the nodules consisted for the most part of purely epithelioid cells, while in the iris, ciliary body, and sclera a mixed type made up of both epithelioid cells and leucocytes predominated. In the nodules of the choroid, giant-cells and leucocytes were present, together with the epithelioid elements. It was quite remarkable that the changes in the retina, choroid, and optic nerve that were brought about by the presence of these tuberculous nodules were, in the main, simply of a mechanical nature. In the case of the retina, the nodules looked like aseptically imbedded foreign bodies replacing normal tissue. In the anterior portion of the eye, however, the changes were, as has been mentioned, of an inflammatory character. It was noted, moreover, that wherever the tuberculous nodules were absent the elements of both the retina and choroid were unchanged.

Apart from the extreme interest attaching to this case from other points of view, the secondary development of tuberculous disease of the conjunctiva in the left eye stamps the case as the only one at present extant of diffuse tuberculosis of the eyeball and its appendages.

With reference to tuberculous disease of the cornea, I have frequently observed that a typical parenchymatous keratitis and, more especially, the disease which goes under the name of sclerosing keratitis, may develop from tuberculous eruptions occurring in the zone of transition between the cornea and sclera, and particularly in the ligamentum pectinatum.

Tuberculous disease of the uveal tract may lead to a parenchymatous keratitis without it being possible to recognize the nodules from external appearances. Not only do clinical observations support me in my contention, but pathologico-anatomical proof exists that parenchymatous keratitis belongs to the picture of tuberculous disease of the eyeball, or rather a parenchymatous keratitis can develop upon the foundation of a tuberculous infection of the eye. As to whether tuberculosis of the cornea may be an independent disease, or is always secondary to a similar affection of the uveal tract (including of course the ligamentum pectinatum), my answer, made upon the basis of my own observations, is that tuberculous disease of the cornea is usually secondary to similar disease in the ligamentum pectinatum, but that the marginal zone of the cornea may be primarily the seat of tuberculous nodules, which later on make their appearance in the cornea itself. Not only is the clinical picture different in these two cases, but within certain limits variations in the pathologico-anatomical condition must necessarily occur.

To illustrate the difference in the clinical picture I adduce the following cases, which I have had under daily observation for a long time:

A girl, seven years old, afflicted with hereditary tuberculosis, was being treated for an inflammation of the left eye. In a short time the right eye became affected. Examination showed circumcorneal injection, nodules in the ligamentum pectinatum above and to the outer side, slight hypopyon, and iritis. Gradually nodules, which rapidly became confluent, made their appearance in all parts of the ligamentum pectinatum. The hypopyon remained almost constant, although varying in height. Soon a parenchymatous keratitis made its appearance, starting in the typical way from the periphery and advancing to the centre of the cornea. The changes in the right eye were almost identical with those just described. The disease ran its course in not quite three months, and was followed by recovery. The ophthalmoscope revealed normal fundi. There were no grounds for suspecting syphilis.

Typical parenchymatous keratitis may be said to occur chiefly after the tuberculous nodules have made their appearance in all parts of the ligamentum pectinatum alternately. More frequently do we have the picture of the so-called sclerosing keratitis, and I will adduce a case which may serve as a type of this variety.

The subject was a servant girl, twenty-three years old, from Lower Franconia. Photophobia and increased lachrymation were present in both eyes, and in the right eye there was slight swelling of the upper lid. In the right eye, too, there were circumcorneal injection and a remarkably large number of deposits, small and large, on the posterior surface of the cornea. Moderate iritis. On the third day a nodule made its appearance on the upper and inner part of the ligamentum pectinatum, and afterwards another distinct from the former appeared a little to the outer side of the vertical meridian of the cornea. A few days later a very diffuse light grayish opacity made its appearance in the deeper part of the cornea just in front of the nodules. A little leash of deep-seated blood-vessels gradually developed at this spot, projecting in from the corneal margin, and at the apex of this leash a yellowish-red nodule could be made out, which lasted for quite a long time. Gradually the yellowish-red color disappeared, giving place to a grayish hue, and the clouded area assumed the shape of an arc with its convexity directed toward the centre of the cornea. The vascular leash gradually disappeared and the whole process ceased, leaving a white opacity projecting like a tongue into the cornea. Seven days after the appearance of the nodules in the ligamentum pectinatum of the right eye, some formed also on the left side and presented the same appearance. In this case too there were numerous and some very large deposits upon Descemet's membrane. The iritis was somewhat more violent. In addition to the nodules in the ligamentum pectinatum two superficial nodules appeared, one right on the corneal border and the other ½ *mm* from the border. These little nodules might readily be confounded with eczema pustules, but their subsequent development precluded any such idea. There was, in fact, no exfoliation of the surface, but on the contrary the nodules advanced slowly to the cornea, and, just as in the other eye, there was to be seen here in a few days in the cornea a deeply situated nodule connected with the site from which it started by a dense, deeply situated leash of vessels. The subsequent course in this eye was the same as in the other. The patient had an hereditary tendency to tuberculosis. The general examination gave no evidence of syphilis or general tuberculosis.

Tuberculous disease of the ligamentum pectinatum, however, shows itself not only in the form of circumscribed nodules, but we may have here more diffuse dirty grayish-red granulation masses which may likewise lead to deep-seated corneal opacities.

The *uveal tract* in its different parts is of all the structures of the eye the most frequent seat of tuberculous disease. In tuberculous iritis the nodules are most frequently seen in the ciliary portion of the iris. We cannot always see such nodules, for they may be present for only a very short time, or they may be situated in the deeper layers of the iris. The intensity of the inflammatory symptoms may vary greatly and is not directly dependent upon the number of the tuberculous nodules. The course of the disease varies too, but most commonly the disease runs a chronic course with occasional exacerbations. Usually there are not many synechiæ, but a very striking feature is the very great number of deposits in Descemet's membrane. Tuberculosis of the iris can also make its appearance as a proliferating granulation-tumor. My own experience here during seven years has taught me that iritis is just as often of tuberculous as of syphilitic origin.

Tuberculosis of the ciliary body shows itself in the form of a chronic inflammation associated with opacities and connective-tissue formation in the vitreous. Occasionally we have numerous hemorrhages into the vitreous body. In other cases of tuberculosis of the ciliary body the sclera may be secondarily involved—a tuberculous nodule of the ciliary body disintegrating and pushing its way through to the sclera so that a tuberculous ulcer is produced in the latter.

Tuberculosis of the choroid is observed under the form of acute miliary tuberculosis and chronic tuberculosis. Ophthalmoscopically we see in the beginning of this disease yellowish-white or rosy-yellow nodules with usually hazy outlines. These nodules sometimes show a slight but distinctly visible projection in the direction of the retina, and soon become surrounded by pigment bodies, especially when they are very prominent. In the middle of these diseased spots are often seen isolated collections of pigment. Hemorrhages are not infrequently seen near by. At a later stage of the disease the pigment-epithelium of the retina over a wide area loses its color, and in part is aggregated into little clumps. The choroid has a dirty yellowish-white color. Within the diseased area or on its borders are often seen round, sharply cut, yellowish-white spots. The choroidal vessels often show perivasculitis. Neuritis optica or even marked atrophy of the optic nerve may be seen. Michel has observed in rare cases tuberculous granulation-tumors of the choroid associated with the appearance of a choroidal or retinal detachment or an abscess under the scleral conjunctiva or in the form of a cheesy abscess in the vitreous.

Tuberculous nodules in the *retina* make their appearance chiefly in the cerebral layer, and in almost all cases the disease here is secondary to tuberculosis of either the uveal tract or optic nerve. Sometimes small yellowish-white elevations appear in the optic papilla and are to be regarded as tuberculous nodules. According to Michel the entire optic papilla may look like a yellowish-red surface, and projecting out into the vitreous body there may be reddish nodules hanging like grapes on whitish threads.

Tuberculosis of the *optic nerve* shows itself most often under the form of an acute or chronic tuberculous meningitis, and the disease may be confined to the sheaths of the optic nerve, without involving the meninges of the brain, or the disease may be transmitted to the sheaths of the nerve from the meninges of the brain. The nerve, as a rule, is studded with tuberculous nodules. If nodules are found in the retina, they will be generally seen also in the optic nerve, and in this case, too, the meninges may be entirely free from tuberculous disease.

A lad fifteen years old had presented for some time the picture of choked disc in his right eye. Later on the same condition showed itself in the left eye. This was followed by atrophic discoloration in the optic papilla. In the case of the left eye there was slight exophthalmus. The autopsy showed that the optic nerve in its course through the orbit was encapsulated in tuberculous granulation tissue, and the same was true to a less extent of its course at the base of the brain. The chiasm and the adjoining parts of the brain were almost completely destroyed by the tuberculous granulation tissue. There was primary tuberculosis of the right temporal bone.

The optic nerve, then, may be the seat of tuberculosis in its entire course. With the ophthalmoscope we find the picture of acute or chronic optic neuritis, or of a primary chronic degenerative atrophy. According to Michel, in those cases where there are masses of tuberculous granulation tissue in the brain, either the cerebral communications of the optic nerve may undergo destruction, or the nerve may be implicated indirectly under the form of choked disc.

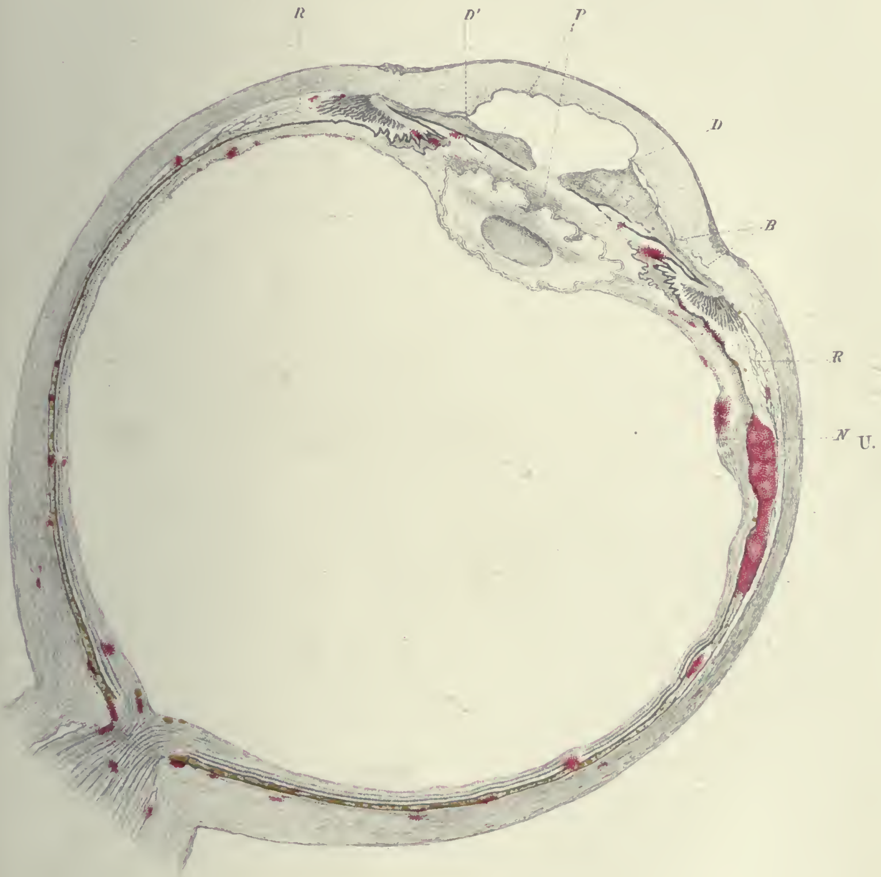
The *nerves*, too, *of the ocular muscles* may suffer in cases of tubercular basilar meningitis, and then paralysis of the muscles ensues. Tuberculous ostitis of bones at the base of the brain may also lead to this result. We also have tuberculosis of the *walls of the orbit*, showing itself in tuberculous ostitis and periostitis, and by the extension of this process to the optic foramen we may possibly get such compression as to cause atrophy of the optic nerve. If the ostitis and periostitis are localized in the orbital margin, cicatricial ectropion may result. Michel has found tuberculous nodules in the *cellular tissue* of the orbit, and is of the opinion that many cases of spontaneous cellulitis in children and young people may be attributed to a tuberculous infection.

Finally I may add that the so-called *blennorrhœa of the lachrymal sac* may be brought about either by tuberculous ulcers of the lachrymal passages or by tuberculous disease of the bony walls lining them, with secondary involvement of the mucous membrane.

Tuberculosis of the eye is most often seen in children and at the age of puberty, though the disease cannot be said to spare those of a riper age.

Explanation of Plate II.

Sagittal section, 5 x. The tuberculous nodules are indicated by red color and dotting. Their number and distribution are diagrammatically drawn on the basis of about fifty sections through the middle of the eye. B. New formation of connective tissue in ant. chamber. P. The same in the papillary space and the posterior chamber. R. Dilatation of the perichoroidal space. N. Proliferation of the retina.



VERLAG VON J. F. BERGMANN, WIESBADEN.
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OXYCEPHALY AND DISEASE OF THE OPTIC NERVE.

BY DR. L. WEISS AND DR. O. BRUGGER.

Translated by Dr. ROBERT L. RANDOLPH, of Baltimore, Md.

(With Plate III. of Vol. XXVIII., German Edition.)

W. M., fourteen years old, was supposed to have had poor vision from earliest youth. At the first glance the fixed look of the prominent eyeballs and the characteristic form of the skull were striking. The pronounced prominence of the eyeballs would lead one to suppose that there existed a high grade of myopia; but on testing his vision this was not found to be the case. Without glasses, vision R E $= \frac{3}{60}$; L E $\frac{5}{60}$. Glasses—especially concave glasses—do not improve vision. Examination with the ophthalmoscope revealed a very low grade of hypermetropia. Without glasses his near vision was equivalent to Jaeger No. 8 at about 6 *cm*, and with convex, 4.0 D, he could see Jaeger No. 5. The extreme boundaries of the field of vision, reckoning from the blind spot as the middle point, were, *temporal*, 60°; *nasal*, 40°–50°. Color sense normal.

The field of fixation, when tested for large objects, proved to be approximately normal. As regards the condition of the muscles, prisms showed for the distance a dynamic divergence of 2°, and for the near, dynamic divergence 18°–20°. When the patient was told to fix an object straight in front of him, the eyes were moved upwards, a phenomenon which either meant that the fovea was not concerned in fixation, or that the visual line passed through the cornea in an abnormal way somewhat below the middle of the cornea. Pupils reacted to light, and the eyes were absolutely free from irritation.

The marked prominence of the eyeballs was measured with the exophthalmometer, and it was found that the apex of the cornea lay 19½ *mm* anterior to the outer rim of the orbit. Ophthalmoscopic examination revealed clear media. The refraction was slightly hypermetropic. The papillæ were conspicuously pale and of a bluish-gray color; and the border of the papilla was surrounded by a light halo, the breadth of which was not everywhere the same. There were no striking changes seen elsewhere in the fundus, except in the macular region in both eyes, where there was to be seen a number of small white spots and points, between which there was increased pigmentation. From this condition a diagnosis was made of postneuritic atrophy of the optic nerve, resulting most probably from a neuritis which had existed in earliest childhood, and to this view of the case pointed the pale gray color of the papilla and the indistinct halo surrounding it, and the little spots in the macular region.

There must not go unmentioned the abnormal course followed by a blood-vessel in the second eye. Right on the papilla a tolerably large arterial branch passes towards the temple and disappears suddenly on the border of the papilla without again emerging in the periphery. And one can convince one's self that this is the case by observing that that portion of the retina belonging to this arterial branch is supplied by a vessel that runs back, leaving the main artery at some distance, and running almost directly backward in a direction opposite to that followed usually by the retinal vessels.

The history amounted to little. At birth the child was delivered with forceps. The father died of softening of the brain.

The shape of the head was remarkable. It was small and very high, and on the top, and at a point corresponding to the position of the large fontanelle, there was to be felt a ledge jutting out like a crest. The peculiar shape of the skull showed itself also in the shape of the orbits, the height of the latter exceeding the breadth. The various head measurements were as follows:

Head circumference, 498 *mm.*
Greatest antero-post. diameter, 177 *mm.*
Greatest transverse diameter, 139 *mm.*
Breadth of forehead below, 105 *mm.*
Distance between cheek-bones, 118 *mm.*
Distance between temporal bones over tragus, 126 *mm.*
Height of face reckoning from the chin, 110 *mm.*
Height of upper portion of face:

a. to the mouth (labial fissure), 72 *mm.*

b. To the lower border of the alveolar process of superior maxilla, 66 *mm.*

Distance from the ear to the external rim of the orbit, R 68, L 69.

Distance from the ear to the middle of the eye when closed, R 92, L 91.

Distance between the two external rims of the orbits, *i. e.*, from external angular process of one orbit to the same point on the opposite side, 94½ *mm.*

Distance from the inner wall of one orbit to that of the other orbit, 30 *mm.*

Height of orbit, R 34, L 33½ *mm.*

Breadth of orbit, I. R 31½, L 31 *mm.*

" " II. R 35, L 34½ *mm.*

Angle of inclination at orbital entrance II.[1] measured with inclination-angle-meter:

$\sphericalangle\ \gamma = (\frac{\alpha - \beta}{2})$ R 15. 5°, L 18°.

Length-breadth index, $\frac{139}{177}$. 100 = 78.00 (mesocephaly).

Upper facial index, $\frac{72}{128}$. 100 = 56.00 (leptoproscopy.)

Orbital indices R, $\frac{34}{31 \cdot 5}$. 100 = 108, L, $\frac{33,5}{31}$. 100 = 108.

With a head of such peculiar shape the supposition was quite natural that the optic-nerve affection as well as the exophthalmus stood in causal relation to the skull deformity. This prominence of the eyeballs was not the result of an abnormally long eyeball, as one sees in high grades of myopia, but was caused by the high position which the eyeballs occupied in the orbit, this position being due to the peculiarly flat build of the orbits.

The first case of this kind reported comes from Michel in the year 1873 (*Archiv für Augenheilkunde*, xiv.). It was Michel's opinion that the hyperostoses of the skull in this case, which were brought to light at the post-mortem examination, had led to a narrowing of the optic foramina, and secondarily to compression and inflammation of the optic nerves. As to the manner in which the neuritis was caused, he thought that the fluid contained in the intravaginal spaces of the optic nerves suffered a retention, and this caused pressure upon the vessels of the optic nerve. In this case choked disc followed by blindness made its appearance in the first year of the child's life. Atrophy of the papilla was the result. The patient, partially demented, died of an intercurrent affection at the age of fifteen years. The skull was a strongly asymmetrical oxycephalic one, with marked hyperostosis and closure of nearly all the sutures.

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[1] *I. e.*, the angle between the plane of the orbital entrance and a transverse plane passing through the inner margins of the two orbits.—See L. Weiss, *Arch. f. Augenheilk.*, xxi., p. 2.

Von Graefe (*Archiv für Oph.*, xii., p. 133) reports a case of double papillo-retinitis followed by total blindness in the case of an eight-year-old boy with an oxycephalic head.

Hirschberg (*Beitraege zur prak. Augenheilk.*, 1876) reports two similar cases where double-sided atrophy of the papillæ existed. In these cases the affection was evidently congenital or at least had started in earliest childhood. The formation of the skull exerted a marked influence on the functions of the brain: in one case congenital dementia, in the other case epilepsy, existed. Hereditary influences in these cases were clearly traceable. In Graefe's case, the mother had epilepsy, the father committed suicide, and of seventeen children sixteen died of convulsions in the earlier years of childhood.

In Hirschberg's cases, the children were subject to convulsions; some died young, and one was an idiot. Later Hirschberg reported seven cases (*ibid.*, January, 1883) of more or less pronounced oxycephaly. In four of these cases Virchow was of the opinion that there existed a premature synostosis of the sagittal suture. In all of them a high grade of amblyopia with narrowing of the field of vision was present and in some cases complete blindness. The bad vision was due to post-neuritic atrophy of the optic nerve.

In the first case reported by Hirschberg, the inflammation had not run its course. At the time the child first came under observation it was five years old and had an exquisite oxycephalic skull. Vision in right eye was good, and in left eye poor. Ophthalmoscope showed regressive choked disc. After nine years, the right eye had become very amblyopic and the left eye totally blind, and in this time the optic nerves had become quite atrophic, the eyeballs prominent, and the lid commissure had an oblique direction.

The child is supposed to have died of convulsions. Virchow thought it probable that it was a case of old meningitis. Hirschberg concluded from his observations that the skull deformity and the optic-nerve affection stood in causal relation with one another, and that both these pathological processes were to be attributed ultimately to an inflammation of the dura mater.

Vossius (*Klinische Monatsblätter für Augenheilkunde*, Jahrg. 1884, p. 172) reported a case of skull deformity associated with optic-nerve atrophy, but the case does not properly belong in this report as it was not one of oxycephaly.

Stood (*ibid.*, p. 248) relates two cases of amaurosis associated with deformity of the skull. One was that of a child four months old where there existed atrophy of the papilla. The other child was seven months old and here the ophthalmoscope revealed nothing abnormal. In both cases the skull was high and there was ossification of most of the sutures and furthermore there was an abnormally small development of the occipital region, and this latter condition by interfering with the development of the central organ of vision, was thought by Stood to explain the blindness in the second case.

At the meeting of the Ophthalmological Congress in Heidelberg, in 1888, Manz called attention to the fact that deviations from the normal diameter in the skull are apt to bring about optic-nerve trouble, as is seen, for instance, in hydrocephalus and microcephalus, and especially true is this of oxycephaly. Of this latter condition Manz reports three cases. All of the patients had attained an advanced age and had been nearly blind from earliest youth, the cause of the blindness being evidently a post-neuritic atrophy. The fact that neuritis had been present was clear, for the border of the papilla looked irregular and washed out. The vessels, and especially the arteries, were thin. The eyeballs were prominent, and showed divergence. The prominence was due to the high location of the eyeballs, and this latter condition was to be attributed to the flatness of the orbit. In two of the cases reported by Manz there existed, just as in my case and in several of Hirschberg's, a well marked sagittal crest. In one of Manz's cases a post-mortem examination was obtained, and, in addition to the changes in the skull, a sarcoma of the right parietal bone was discovered. Two years before his death the patient had a marked bulging forward of the right parietal region, due, no doubt, to the sarcoma. The right eye at this time was completely blind, and the ophthalmoscope revealed post-neuritic atrophy. The left eye showed choked disc with beginning atrophy and defects in the visual field below to the inner and outer sides. The choked disc disappeared in the course of time, and the vision returned to the normal. At the autopsy, in addition to the sarcoma, there was found a pronounced development of all the prominences at the base of the skull. The roof of the left orbit rose up high, while the right was depressed. The foramina, through which the nerves and vessels passed, were enlarged, and had sharpened borders. The changes must have existed since earliest youth, for nothing pointed to a recent bone affection. Manz concluded that there had been present in uterine life a condition of ostitis associated with pachymeningitis and leptomeningitis, all of which affections led to the changes in the bones, with the subsequent optic-nerve atrophy. This explanation excluded the possibility of stenosis of the optic foramen, though in the last case both optic nerves showed a marked contraction at the points adjacent to the optic foramina.

Oeller observed optic-nerve atrophy in the case of two brothers with oxycephaly. The brothers were strikingly alike, and the ophthalmoscopic picture in both cases presented a remarkable resemblance. The upper and inner border of the papilla showed very indistinct outlines. At the lower border of the papilla there was a lighter area triangular in shape, and with indistinct outlines, and at this point the choroidal vessels were plainly seen. We are right, then, in assuming that a relation exists between deformities of the skull and affections of the optic nerve, though we cannot say at what stage the disease of the optic nerve makes its appearanee. In those cases where autopsies were held, the disease had run its course. Autopsy cases where it was possible to make out unquestionably a causal relation between the two affections,—fresh cases in other words,—have not yet been met with. Furthermore, absolute proof is wanting of a connection existing between the disease of the optic nerve and the disease of the central organ. So, also, we cannot explain what relation the disease of the central organ bears to the skull deformity. Thinking it possible that certain changes in the bones of the skull might be made out, changes which were of moment in their bearing upon the optic-nerve trouble, I have examined all the oxycephalic

TABLE I.

Head Measurements.	Oxycephalic Skulls.				Average Measurements.	Average Measurm'ts of 100 Normal Skulls.
	No. 2.	No. 5.	No. 13.	No. 15.		
Horizontal circumference...	473	483	474	482	478	
Antero-post. diameter.....	142	165	158	160	156.2	172.69
Greatest lateral diameter..	131	133	135	140	134.8	143.41
Height of upper part of face	67	78	69	72	71.5	67.1
Distance between cheek-bones, according to Virchow	116	123	118	117	118.5	122
Greatest breadth between cheek-bones...........	*	130	123	121	124.7	129.1
Lower breadth of forehead.	97	94	94	83	88.75	97.77
Distance between the meat. aud.	100	97	96.5	98	97.9	104.5
Height of skull...........	137	140	146	149	143	131.6
Base of skull.............	93	91	92	89	91.25	98
Distance from the middle of the meat. aud. to the ext. orbital border. R.	67	65	65	58		
L.	67	67	65	57	63.9	69.1
Distance from the external orbital border to the middle of the dorsum sellæ. R.	70	64	67	60		
L.	70	63	66.5	60	65	71.42
Distance between the mastoid processes..........	106	90	98	105	99.8	
Distance of the root of the nose from middle of dorsum sellæ..............	67.5	60	64	55	61.6	
Distance of the root of the nose to the ext. orbital border. R.	51	52	48	47		
L.	52	52	47	47	49.5	
Distance of the dorsum sellæ to optic foramen..	about 20	21	14	18	18.25	

* Cheek-bone broken off.

skulls in the Heidelberg Anatomical Institute. Special attention was directed to the anatomical conditions or relations of the orbits. The result of these measurements is given in the following table. The measurements of 100 normal skulls are given in the same table for purposes of comparison.

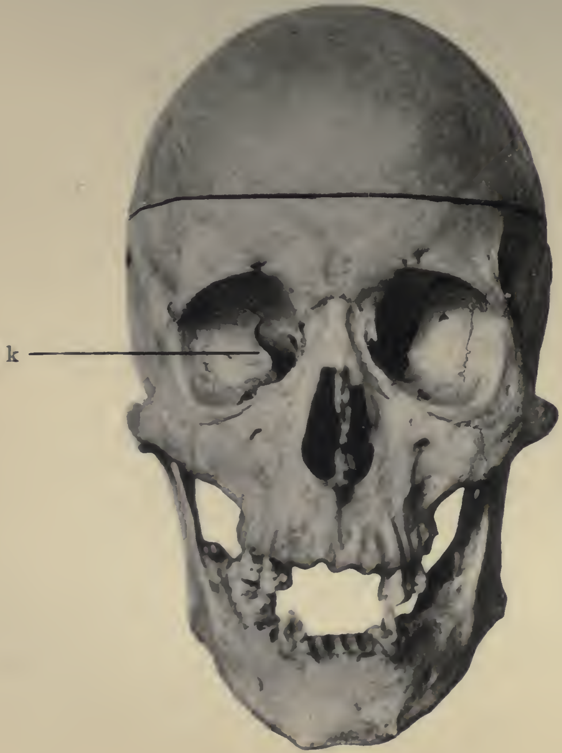
From an examination of these tables, one is particularly

TABLE II.

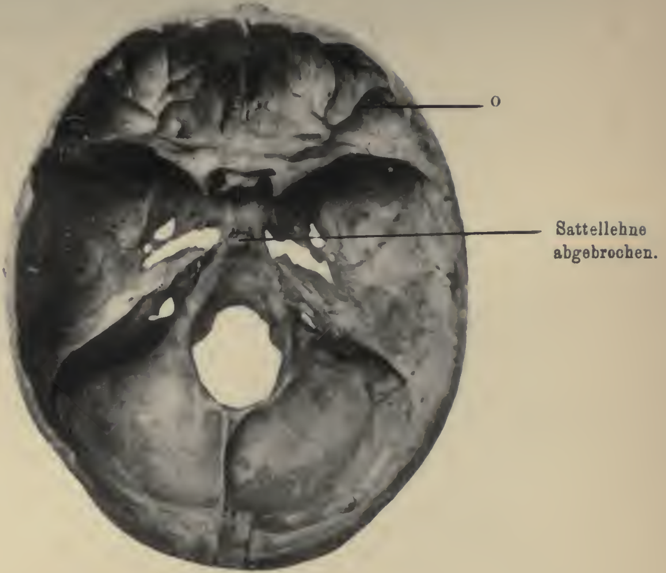
Orbital Measurements.	Oxycephalic Skulls.				Average Measurements.	Average Meas'm'ts of 100 Normal Skulls.
	No. 2.	No. 5.	No. 13.	No. 15.		
Height of orbit at entrance R.	34	37	36.5	39	36 55	34.3
Height of orbit at entrance L.	34	36.5	36.5	39		
Breadth I R.	36	36.5	35.5	35	35.5	36.52
Breadth I L.	35.5	36.5	35.5	34		
Breadth II R.	37.5	39.5	39	38.5	38.4	39.7
Breadth II L.	38	39.5	39	36.5		
Great oblique diameter R.	38	41.5	41.5	41.5	40.8	41.46
Great oblique diameter L.	38	41	44	41.5		
Small oblique diameter R.	34.5	36.5	34	34	34.6	34.35
Small oblique diameter L.	34.5	36	34	34		
Distance between the inner orbital walls	27.5	22.5	21	21	23	27
Distance between external orbital borders	99	93.5	96	88.5	94.25	97.3
Distance between the foramen optic.	21.5	15	16.5	21	18.5	21.58
Distance between the foramen optic. orbital	27.5	26	25.5	26.5	26.4	30.3
Depth of the orbits: Inner wall R.	36	27.5	29	30	30.4	40.3
Depth of the orbits: Inner wall L.	36	26	29	30		
Depth of the orbits: Outer wall R.	45	39	42	39	41.1	47.4
Depth of the orbits: Outer wall L.	45	38	42	39		
Depth of the orbits: Perpendicular to the orbital entrance I, R.	34	25.5	28	29.5	28.4	39.05
Depth of the orbits: Perpendicular to the orbital entrance I, L.	34	25	28	29.5		
Distance of the point of intersection of this perpendicular with the plane of the orbital entrance from the inner wall. R.	8	8	5	8.5	7.45	12.54
Distance of the point of intersection of this perpendicular with the plane of the orbital entrance from the inner wall. L.	8	8	5	9		
Cubic contents of the orbits.	25	21	27	21.5	23.8	29.74
Angle of inclinat'n of the orbit'l ent'nce I. $\sphericalangle \gamma = (\alpha - \frac{\beta}{2})$ R.	13	12.5	11	4.5	10.25	
Angle of inclinat'n of the orbit'l ent'nce I. $\sphericalangle \gamma = (\alpha - \frac{\beta}{2})$ L.	12.75	12.5	12	4	10.31	
Angle of inclinat'n of the orbit'l entrance II $\sphericalangle \gamma = (\alpha - \frac{\beta}{2})$ R.	16	13.5	11.25	11.25	13.0	15.8
Angle of inclinat'n of the orbit'l entrance II $\sphericalangle \gamma = (\alpha - \frac{\beta}{2})$ L.	14	13.5	13	11.5	12.75	

struck by the extraordinary development of the height measurements in oxycephaly at the expense of the antero-posterior diameters, which remain under the medium. The transverse diameters do not differ greatly from the normal. As regards the orbit measurements, we notice marked deviations from the normal. First of all, we notice that the height almost always exceeds the breadth. The orbital entrance does not, as is usual, present the transverse oval shape, but the oval is more or less perpendicular. The structure of the inside of the orbit differs greatly from that seen in the normal orbit. The orbits in oxycephaly are much shallower than the normal orbits—shallower to the extent of 10 *mm.* The cubic contents of the orbits are, on an average, 6 cubic *cm* below the normal. The position of the great wing of the sphenoid contributes more than anything else to the smallness of the space within the orbit. The wing has a strong frontal direction, and instead of forming the side, goes to form the posterior wall of the orbit, so that the latter is changed into a blunt cone of very little height. Generally the wing of the sphenoid pushes forward somewhat over the other bones (see Plate III., Fig. 1, on the left of the drawing), especially on the median side, and the latter consequently appears shortened. The superior orbital fissure is generally short and broad, and forms approximately a triangle with the base below. This abnormal construction of the orbit easily explains the prominence of the eyeballs seen in persons having oxycephalic skulls. Corresponding to the great height of the orbit, one will observe in all oxycephalic skulls that the roof of the orbit is arched unusually high, and on the inner surface will be seen marked ruts and prominences. Between the two high arched orbital roofs the ethmoidal plate forms a deep, broad valley (Plate III., Fig. 2). In none of the cases was the optic foramen abnormally narrow. What was remarkable in all these cases was a pronounced lengthening and pushing forward of the clinoid process. This peculiar anatomical condition leads one to believe that at a certain time in earliest youth, as a result of the abnormality in the sella turcica, pressure was exerted at this point on the chiasm, and hence the optic-nerve affection. Worthy of note is the fact that as a general thing these oxycephalic skulls have very thin, delicate bones. A condition which is characteristic of oxycephaly is ossification of the coronary suture. The sagittal suture is often ossified. In some cases supernumerary bones were present, especially in the region of the occipital bone. The cubic contents of the skull were not abnormally small and amounted on an average to 1400–1500 cubic *cm.* In none of our cases was there evidence of a pre-existing bone disease. The bones were smooth and showed no rough spots or pathological changes whatever. The accompanying photographs of skull (oxycephalic) No. 5 (see Plate III.) illustrate very clearly what has just been said.

1.



2.



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HYALINE AND AMYLOID DEGENERATION, WITH CALCAREOUS DEGENERATION AND OSSIFICATION IN AN OCULAR MUSCLE AFTER AN INJURY.

By Dr. OSCAR BRUGGER.

(From the Eye-Clinic of Dr. L. Weiss.)

Translated by Dr. C. Zimmermann, Milwaukee, Wis.

(With two lithographic Plates, XV. and XVI. of vol. xxviii. of German edition.)

Pauline F., æt. fifty-seven, who was admitted to the eye-clinic of Dr. C. Tscheppe at Constanz, stated that eighteen years ago she was struck in the right eye by the horn of a cow, with immediately following profuse bleeding and swelling. Two years ago a physician, who treated her for a muscular paralysis of this eye, discovered a tumor on the eye. It could not be ascertained, whether there had been a deviation of this eye right after the accident, but it seems very probable, especially as the minister of the village several years ago had called her attention to this fact.

Status on admission: The right eye displaced downward and forward, covered by the upper lid, which was in a state of ptosis. V = 4/12. The cause of the protrusion was a tumor at the region of the superior rectus, about as thick as a finger, commencing at the muscular insertion, and extending backwards. It was solid and hard, and appeared as bone on puncture with a needle. It was movable, and not adherent to the globe or the orbit.

June 22, 1891.—Removal of the Tumor. After incision of the conjunctiva the tendon of the superior rectus was visible. This was severed from the eyeball, and pulled forward, so that the tumor could be dissected all around. Its posterior portion was not ossified. The tumor came away entirely by removing the origin of the ossified muscle with an elevatorium. There was no active mobility of the eyeball. The remnant of the tendinous insertion of the sup. rect. was stitched to the orbital fascia. The globe looked now directly forward. The ptosis gradually improved, so that the lid could be lifted above the corneal centre. V = 4/12, as before. Twenty-six days after the operation the patient was discharged cured.

The tumor had the shape of the muscle, but was wider (15 *mm*), and thicker (9 *mm*). It was ensheathed by thickened perimysium, and showed no traces of injury or attachments. The surface was uneven from granules up to 2 *mm* diameter, translucent like wax. Fig. 1 shows a sagittal section of the tumor. The chief mass consists of a network of connective tissue, in which the yellowish granules are imbedded. The distal end contains apparently normal muscular fibres. The larger granules especially are pretty loose, and can be easily enucleated. The smaller ones are mostly round, and show in a section concentric formations, some even a central lumen. A covering membrane can not be isolated by preparation, but its existence becomes very likely from the appearance of nuclei on the surface after treating the granules with acetic acid. The larger granules are mostly irregular and hard, so that they cannot be cut by a knife, without being crushed. Acids cause development of CO_2, and on adding sulphuric acid crystals of plaster of Paris form. They thus undoubtedly contain carbonate of lime. After decalcification only a few nuclei or their remnants are to be seen, which are not dissolved in alcohol and ether, so that their fatty or cholesterine nature can be excluded. On some granules bone corpuscles with nuclei could be observed, before and after decalcification, but the chief mass of the tumor did not consist of bone. Sagittal sections of a piece of the tumor, hardened in alcohol and decalcified by nitric acid, were stained with different methods, of which Fig. 2 gives a view.

The apparently homogeneous granules of waxy lustre are imbedded in the muscular substance. The smallest are as large as one or a few cells; the largest have a diameter of several *mm*. They are undoubtedly produced by the blood-vessels. On several places within the granules, in round sections exactly in the centre, traces of a former lumen or a regular vessel, filled with blood, can be seen. This is a very important point, so that I have illustrated it in Fig. 3. It represents an artery of medium size. The lumen, filled with blood corpuscles, is surrounded by a stratum of circular muscular fibres, with nuclei transversely placed, followed by a stratum of longitudinal muscular fibres, with nuclei appearing roundish. The latter at one place is enlarged and granulated, presumably in consequence of degeneration of the vascular wall. The artery proper is ensheathed by a voluminous, apparently homogeneous mass, with concentric stratification. In this occasional roundish cells are discernible similar to bone corpuscles with nuclei, and numerous clefts and fissures, apparently due to the action of alcohol. A closer examination reveals these cells in most of the granules, even in those not in connection with the blood-vessels.

Another peculiarity of the new formations lies in their reaction to staining. Fig. 3 shows staining with picrolithioncarmin; the periphery of the granules intensely yellow; in the central portions the carmin-staining predominates, though faintly. Hæmatoxylin stains the centre intensely blue, or hardly at all. From their appearance and condition the new formation might be considered as products of hyaline or amyloid degeneration, and to prove this, a few sections were treated with a solution of jodine-jodide of potassium and methylviolet. Some portions of the masses turned intensely brown and light-reddish respectively, the rest straw-yellow and blue respectively. Thus amyloid and hyaline degeneration occurred simultaneously. The hyaline masses showed also the characteristics of hyaline, described by von Recklinghausen, to be mentioned later on.

The muscular fibres are separated by hypertrophic perimysium, and in some places appear imbedded in regularly arranged loose connective tissue. Quite a number, especially in the posterior portion of the tumor look perfectly normal and transversely striped. Towards the tumor proper they become more scarce, and are supplanted by swollen hyaline —degenerated fibres without transverse striation. Besides, there are fibres in which the muscular substance is entirely obliterated, the empty perimysial sheath being all that is left. Some fibres show all these different stages. In the chief mass of the tumor, formed by the hyaline and the amyloid masses mentioned above, no muscular fibres are found, except occasionally an isolated piece of well preserved muscle may be met with between the hyaline masses. The connective tissue between the muscular fibres, the original perimysium, is hypertrophied, and forms a pretty thick and dense coat around these masses. The stroma of the tumor consists of reticular connective tissue, especially in the neighborhood of the hyaline masses. The intermuscular tissue abounds with nucleated cells, large connective-tissue cells, with considerable infiltration with round cells. It is very vascular, especially between the single muscular fibres, and even more so around the hyaline masses, where many distended capillaries are seen. They sometimes expand into large cavities, filled with blood, between the hyaline masses, and are surrounded by connective tissue with numerous cells. The walls of these apparently newly formed vessels mostly consist only of one layer of endothelia, and the stroma resembles granulation tissue. The whole seems to be of inflammatory origin.

Besides the hyaline and calcareous degeneration there was a regular ossification in the muscle (Fig. 6), an interstitial ossifying myositis, starting from the perimysium. The bone forms small, irregularly defined trabeculæ between the hyaline masses. It shows bone corpuscles with cells, no Haversian canals, but larger spaces, filled partly with fat, partly with blood and numerous cells, apparently medullary cavities. The trabeculæ are surrounded by vascularized embryonal tissue, here and there nucleated giant-cells and osteoblasts, covering the surface of the bone like cylindrical epithelium. Von Graefe (*Zehender's klin. Mon.*, 1863) and Saemisch (*Graefe-Saemisch*, vol. iv.) observed ossification in the conjunctiva, von Hippel (*v. Graefe's Arch.*, vol. xxv.) and Kubli in amyloid degeneration of the conjunctiva.

According to von Recklinghausen (*Hand-book of General Pathology of Circulation and Nutrition*), the amyloid, mucine, and hyaline substances, produced by the cell-protoplasma, are not soluble in the juices of the tissues; only swell by imbibition, like colloids. The hyaline is an albumen, which may be intensely stained by eosin, carmine, picrocarmine, and acid-fuchsin. It is homogeneous and refracts the light strongly, is very little changed by acids, in its resistance to alcohol, water, ammonia, and acids like amyloid, without giving its reaction to iodine. Its formation consists in a coalition of the contents of approximate cells. Von Recklinghausen advanced the hypothesis that, in the formation of amyloid, homogeneous material, originating from the cells of an organ, swells in the juice of the tissue like mucus, and forms lumps, bands, nets, membranes, and tubes. Ziegler, however, places the origin of amyloid chiefly in the circulating albumen, which the tissue cells cannot normally dispose of, so that it remains, and becomes modified and transformed into amyloid. According to Ziegler the hyaline formations are the result of congelation of a fluid impregnating the vascular walls. Probably leucocytes and blood-plaques may furnish the material for the hyaline substance, and, according to Oeller (*Virchow's Archiv*, vol. lxxxvi.), red blood-corpuscles also.

The condition in our case may be explained by both theories. At some places the hyaline degeneration had its chief seat in the adventitia; at others it had started from the intima leading to obliteration of the vascular lumen, by accumulation of hyaline masses. In the former case the hyaline masses ensheathed the vessel. Frequently both processes were combined, so that large masses without structure were produced, with abolition of lumen and walls of the vessels. Besides the hyaline degeneration of the blood-vessels and the hyaline transformation of the muscular fibres, there is another matrix, from which the new formation may arise. In nearly all preparations small hyaline granules with nucleated covering membranes are seen in the inflammatory connective tissue exuberant with cells. They look like amyloid granules, as described and illustrated by Leber (*von Graefe's Arch.*, vol. xix.) in amyloid degeneration of the conjunctiva. The smallest of these granules are not much larger than an ordinary cell and have mostly one nucleus. All the large masses mentioned show this nucleated covering, which Leber and others found on the amyloid granules. Leber believes that these granules form by the transformation of a free cell into amyloid substance, except a superficial portion, or that the cell produces such a substance, spreading at the same time as a covering membrane around the secretion. The nuclei in fresh preparations became more marked by acetic acid or picrolithioncarmin. By the latter staining the hyaline mass turns yellow, the nuclei beautifully red (Fig. 5). The nucleated coat could not be dissected from the hyaline granules. They only differ from Leber's amyloid granules by the absence of the amyloid reaction.

Nevertheless the process seems analogous, as, according to a widely spread opinion, the amyloid degeneration is always preceded by a stage of hyaline degeneration. This is Raehlmann's view in regard to the amyloid degeneration of the eyelids (these ARCHIVES, vols. x. and xi.).

In the latest edition of Ziegler's *Text-book of General Pathological Anatomy* it is held that undoubtedly the hyaline formation is related to the amyloid degeneration, as a portion of the hyaline masses can be converted into amyloid, so that combinations of both kinds of degeneration may occur. Ziegler mentions (*l. c.*, p. 148) a case of amyloid degeneration of the peritoneum, in which the central portions of the large foci, not their periphery nor the smaller foci, gave jodine reaction. The same was observed in our case.

As mentioned above, the central portions of the new formations gave marked amyloid reaction, viz., the apparently oldest, which were degenerated first, turned brown with jodine, red with methylviolet. Where a vascular lumen remained the strata next to the vessel gave amyloid reaction, whereas the more peripheral, *i. e.*, more recent portions, showed hyaline formations. Whether and how far the amyloid degeneration was veiled by the extensive cal-

Senkrechter Durchschnitt der Geschwulst.
Makroskopisches Bild.

Fig. 1.

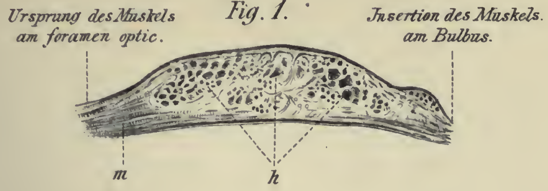
Ursprung des Muskels am foramen optic.
Insertion des Muskels. am Bulbus.
m
h

Fig. 2.

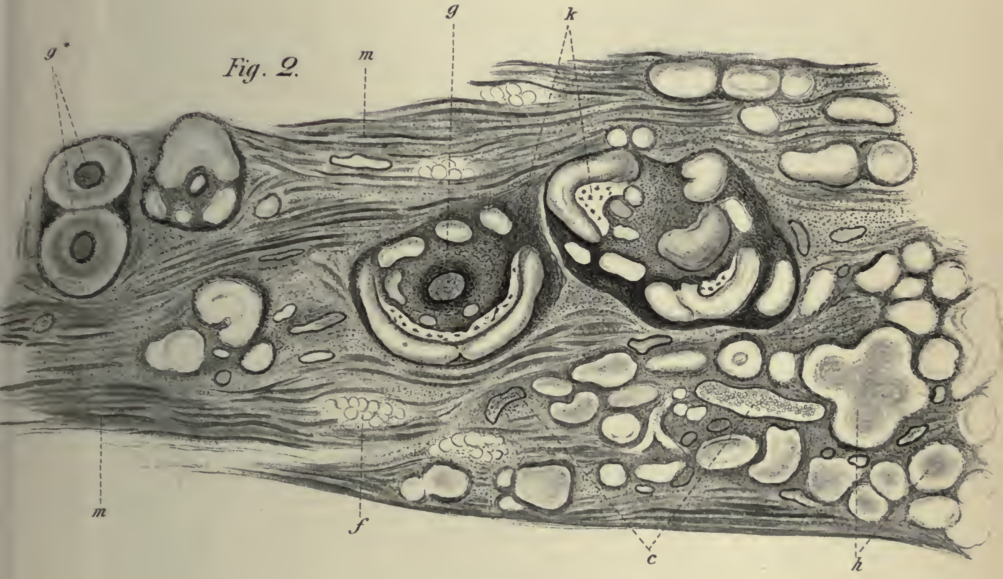

Fig. 6.

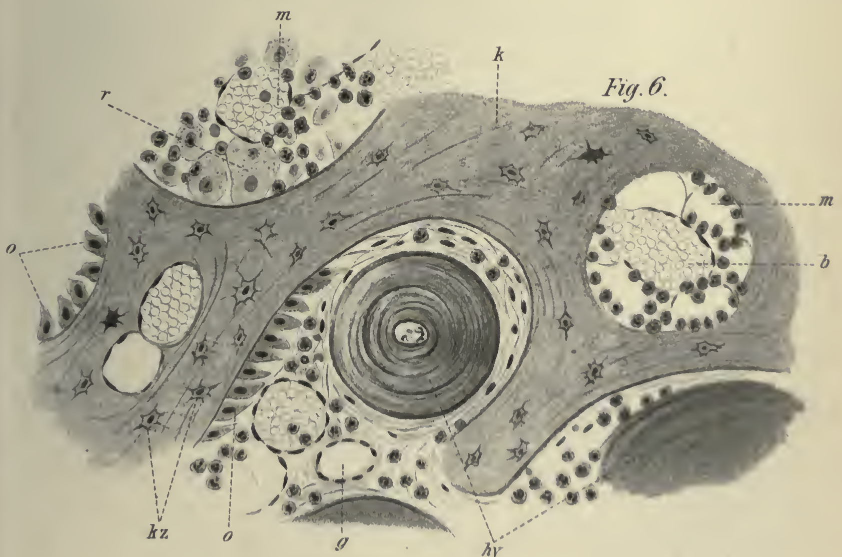

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Fig. 3

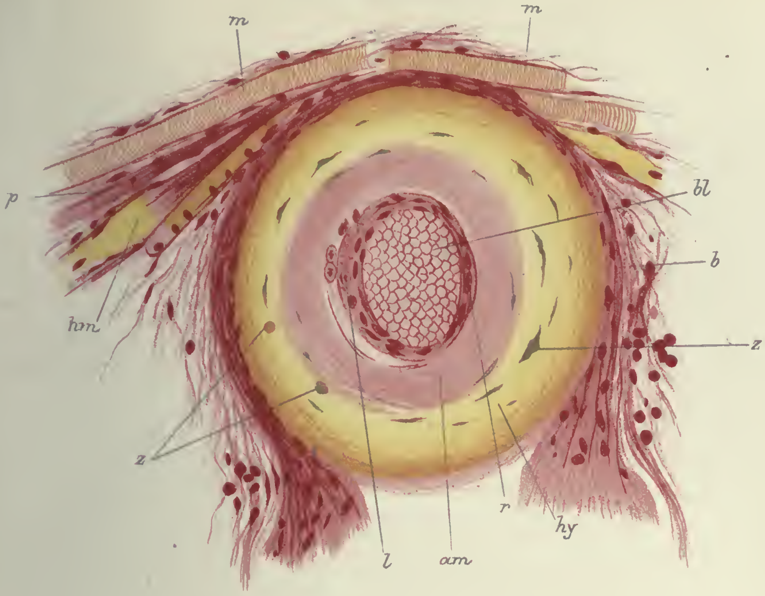


Fig. 4

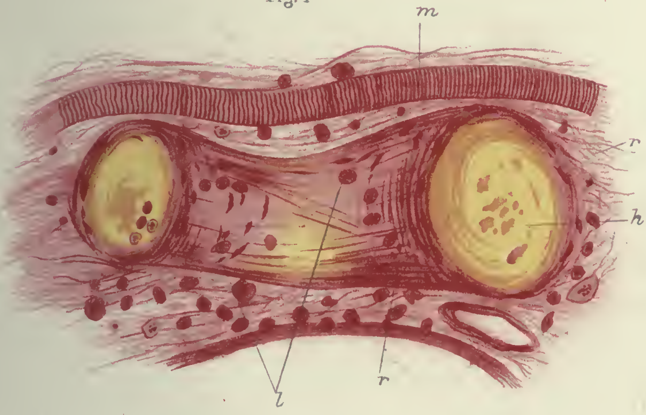
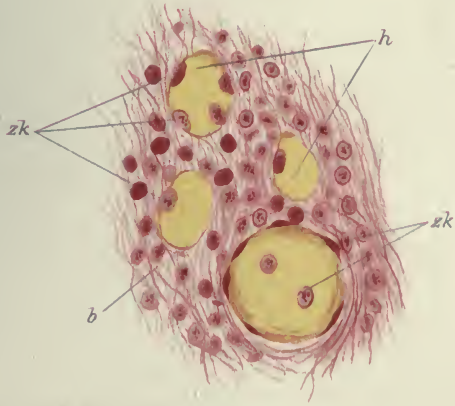


Fig. 5



IVERSITÄTSDRUCKEREI VON H. STÜRTZ, WÜRZBURG. VERLAG VON J. F. BERGMANN, WIESBADEN.

careous degeneration could not be ascertained. In a pathological view hyaline formation with calcification is identical with psammoma. All larger masses were calcareous and developed CO_2 with acids. Only the smallest granules, the coverings of which had only few nuclei, and not being larger than a few cells, were not calcareous. Kubli (these ARCHIVES, xi., p. 149) compiled all published cases of amyloid degeneration of the eyelids, and came to the conclusion that four stages in the development of amyloid tumors of the conjunctiva are to be distinguished: (1) adenoid vegetation in subconjunctival tissue; (2) changes taken as hyaline degeneration by Raehlmann (*l. c.*); (3) marked amyloid degeneration; (4) calcification and ossification. According to this classification our tumor was in a very far advanced stage, the last one. But, as the description shows, there were numerous transitions between all four stages.

From the results of our investigations we met with an extraordinary rare occurrence. Similar changes of an ocular muscle have not yet been described, as far as I could find. Undoubtedly the severe injury created an inflammatory condition in the muscle, resulting, in the course of many years, in the changes described. The orbit being intact it could not be supposed that a bone chip, separated from the orbital roof, might have been implanted into the muscle, giving rise to ossification. And then the bone in the tumor was not a large piece, but consisted of small thin bands and trabeculæ scattered all over the tumor and not continuous.

Explanation of Plates XV. and XVI.

FIG. 1.—Sagittal section of the muscle (macroscopic view). *m*, muscular fibres; *h*, hyaline, and amyloid portion of tumor with ossified and calcareous places.

FIG. 2.—Section through the posterior portion of tumor at the transition into the muscle; *m*, muscular fibres; *f*, fat; *c*, capillaries; *g*, larger vessels; *g**, medium-sized arteries surrounded by hyaline masses; *k*, bone.

FIG. 3.—One of the vessels of Fig. 2 marked with *g**; *m*, normal muscular fibre; *hm*, muscular fibre in hyaline degeneration; *p*, empty perimysium sheath; *am*, amyloid substance (this portion

in a section through the same vessel stained intensely brown by iodine); *hy*, hyaline substance; *z*, remnants of cells in the hyaline substance.

r, Circular muscular coat of the arterial wall.

l, Longitudinal muscular coat of the arterial wall.

bl, Lumen of artery, filled with blood; *b*, covering membrane of the hyaline mass.

FIG. 4.—Vessel cut longitudinally; *m*, normal muscular fibre.

r, Circular muscular coat of the vessel.

l, Nuclei of longitudinal muscular coat of the vessel.

h, Lumen of the vessels, filled with hyaline masses.

FIG. 5.—Smallest hyaline granules; *h*, hyaline granules; *zk*, cell-nuclei on the hyaline granules; *b*, reticulum of connective tissue with intense cellular infiltration.

FIG. 6.—Portion of the tumor with bone; *k*, bone; *kz*, bone corpuscles containing cells; *o*, osteoblasts; *r*, giant-cells; *m*, medullar cavities filled with blood (*b*), fat, and various cells; *g*, blood-vessels; *hy*, hyaline masses with marked concentric stratification.

MICROPHTHALMUS WITH ORBITO-PALPEBRAL CYST.

By Dr. H. BECKER,

ASSISTANT TO THE UNIVERSITY EYE CLINIC AT MARBURG.

Abstract from *Arch. f. Augenh.*, vol. xxviii., p. 81, etc., by Dr. Ward A. Holden.

(With Plate IV., Vol. XXVIII., Germ. Ed.)

THROUGH the kindness of Prof. Schöler and Prof. Uhthoff, I am enabled to report the following case:

May 2, 1891, a male infant, aged six months, was brought to Prof. Schöler's clinic with a tumor lying beneath the conjunctiva of the lower lid, filling the entire palpebral fissure and pressing the lower lid forward. The tumor was translucent and fluctuating, and was as large as a pigeon's egg. When the lids were opened a small eyeball with an opaque cornea could be seen. The child was otherwise well formed and healthy.

In the removal the cyst collapsed and a clear yellow serous fluid escaped. From the posterior inferior surface of the eyeball a small conical process projected into the lumen of the cyst, which was connected with the eyeball. The eyeball measured 12 *mm* x 10 *mm*, and the cyst 8 *mm* x 12 *mm*, with walls 1 *mm* thick.

The eyeball and cyst were cut in serial sections and stained with hematoxylin-eosin or carmine.

Macroscopically the ball was not spherical, having an ectasia of the sclera just above the cornea. Posteriorly a defect in the sclera extended back to the optic nerve, and through this defect a cone of tissue passed into the cyst.

The corneal stroma was infiltrated with leucocytes anteriorly, and contained blood-vessels. The iris lay on the posterior surface of the cornea for the most part. The pupil was excentric, and contained a persistent membrane. The lens was partially cataractous, and was relatively very large, nearly filling the interior of the ball, and displacing the ciliary processes. No zonula fibres could be found. Choroid and pigment-epithelium were normal, and were everywhere present, extending a short distance through the scleral defect. A finely fibrous tissue, rich in cells and poor in vessels, extended from the interior of the ball 3 *mm* into the interior of the cyst in a conical form, then continued over the inner surface of the cyst as the inner layer of the cyst wall (Fig. 1, *i*, Pl. IV.), the outer fibrous layer being continuous with the sclera (Fig. 1, *a*, Pl. IV.). The inner layer of the cyst wall began just behind the scleral cleft as a layer of cylindrical cells with long fibres (Fig. 2, *c*, Pl. IV.), which it would be difficult to consider mis-shaped rods and cones. This layer of epithelium had many folds, which in the sections often resembled glandular depressions (Fig. 3, Pl. IV.). In other parts the single stratum of cells was thickened into a fibrous and cellular network resembling gliomatous tissue. The retina within the ball was normally developed at some points, particularly in the scleral ectasia anteriorly, while at others it consisted only of a single layer of cells.

In this case there was a communication between eyeball and cyst through a cleft in the former, and through this communication a fibrous tissue rich in cells passed from the interior of the ball into the interior of the cyst. Since this tissue was accompanied by pigment epithelium it is to be considered an analogue of the retina. The inner layer of the cyst wall therefore has arisen from the retina of the ball, and the whole cyst stands in genetic relation to the ball. Since all the constituents of the normal ball are present except the zonula, the cyst must have developed at a late stage when the lens was already in the interior of the eye.

Until recently Arlt's theory of the formation of these cysts was the one generally accepted. He believed that at the time when the secondary ocular vesicle has formed but the fœtal cleft is still open, an increase in intraocular tension distends the ocular walls near the cleft, and the cyst arises

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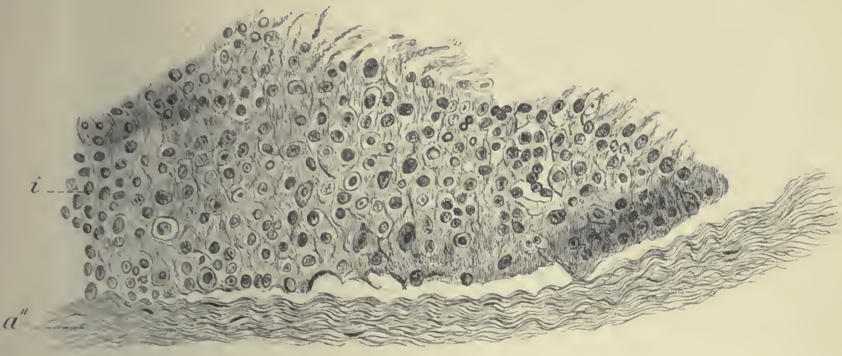

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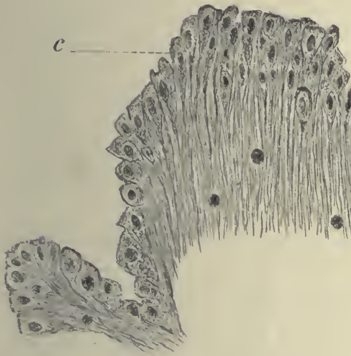

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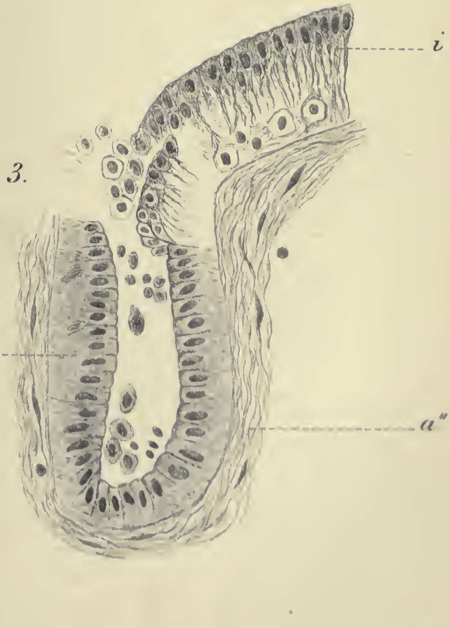

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as a simple ectasia. But in cases examined later the retinal layer lining the cyst was always found to be inverted, and Kundrat advanced the theory that the wall of the primary ocular vesicle pushed out into the mesodermic tissue to form the cyst. This view was taken later by Czermak, Lang, and Mitvalsky (in whose paper in these ARCHIVES, vol. xxii., p. 355, a discussion of the different theories and an abstract of the literature may be found.—*Trans.*). In cases in which the lens is not present this theory of the excessive development of some portion of the primary ocular vesicle is readily understood ; but for the cases in which a fully formed lens is present, the secondary ocular vesicle must have formed. Mitvalsky avoids the difficulty by supposing that the primary vesicle is involuted anteriorly so that the lens may be formed, but that involution does not take place posteriorly near the nerve, and here a cyst develops. This would seem to have taken place in the present case.

Explanation of the Figures.

FIG. 1. (Pl. IV.).—*i*, inner wall of cyst ; *a*, outer wall of cyst.

FIG. 2.—Cylindrical epithelium of the inner wall of the cyst, with long, fibrous processes.

FIG. 3.—*i*, cylindrical epithelium forming a sac-like depression ; *a*, outer wall of cyst.

TUBERCULOSIS OF THE LACHRYMAL GLAND.

By Dr. K. L. BAAS,

PRIVATDOCENT AND ASSISTANT AT THE UNIVERSITY CLINIC AT FREIBURG.

Abstract from *Archiv f. Augenheilk.*, xxviii., 1, by A. Duane, M. D.

(With Plate V., of Vol. XXVIII., German Edition.)

UNTIL very recently practically no data in regard to tuberculosis of the lachrymal gland have been forthcoming. A year ago, however, F. de Lapersonne[1] published a case of it, and Dr. L. Müller described two cases of what he regarded as primary tuberculosis, observed at Prof. Fuchs's clinic in Vienna. These two authors, so far, are the only ones that have described this new form of disease.

De Lapersonne's case was that of a single woman, thirty-two years of age, who had no hereditary tendency to tuberculosis, but who some years before had been treated for a "chronic bronchitis," which was attended with hæmoptysis, fever, and rapid emaciation. Subsequently all these symptoms disappeared, and auscultation revealed only a slightly roughened respiration at the apex of the left lung.

For about three months previous to the time when de Lapersonne examined her the patient had noticed a slight drooping of the right upper lid, and later on a swelling which formed in the external and upper angle of the orbit. Palpation revealed the presence of a subcutaneous tumor about the size of an almond, of fibrous consistence, and irregular surface. This was regarded as an adeno-sarcoma, but the anatomical examination by Prof. Hermann gave the following surprising result:

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[1] *Archives d' Ophtalm.*, vol. xii., 1892.

On section are seen small, rounded, lardaceous lobules, united to one another by delicate connective tissue. But little of the normal gland tissue is any longer visible, and the tissue appears very dense, and is more opaque than it should be. Staining with picrocarmin clearly differentiated the healthy from the diseased tissue. The gland tissue that was still present was infiltrated with numerous cells. In those spots which are more affected, the gland tissue is pressed apart by dense masses of embryonal tissue, in which islands of large cells, owing to the way in which they are stained, stand out very prominently. These cells have an ovoid, vesicle-like nucleus. In the parts most affected the original tissue is almost completely replaced by new-formed tissue of this character containing epithelioid cells. Giant-cells are found but sparingly; but, on the other hand, the epithelioid cells are arranged to form perfect tuberculous nodules, which develop probably by way of the lymph-channels. Staining of the sections by Ehrlich's method did not reveal the presence of any tubercle-bacilli.

From the result of this examination de Lapersonne made the diagnosis of tuberculosis of the lachrymal gland, although with the reservation which he felt imposed upon him by the failure to find bacilli, and the fact that no attempts had been made to inoculate animals with the morbid material.

In Müller's first case,[1] that of a girl fourteen years of age, the affection of the right eye began four years before she presented herself for examination. There was found, beneath the outer half of the reddened and swollen upper lid, a new growth which produced a nodular swelling of the conjunctiva, and which was continued backward. As a relapse occurred a month after the removal of the growth, a second operation was performed upon the tumor, which was hard and lobulated. In the portion first extirpated a distinct lobular structure was made out; and while in one portion the lobules of the lachrymal gland showed only a moderately developed infiltration with small cells, in another portion these lobules were "replaced partly or entirely by typical tuberculous granulation tissue; the tuberculous tissue was surrounded and shut off from other parts by a capsule, which was the non-affected, inter-acinous connective tissue." Giant-cells were few and, comparatively speaking, but slightly developed. Tubercle bacilli were found in very great numbers.

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[1] L. Müller, "Ueber primäre Tuberculose der Thränendrüse," *Festschrift f. Billroth*, p. 144.

The second case was that of a man, forty years of age. Here too, a hard, deeply situated tumor was found; and a recurrent growth that formed was almost as hard as cartilage. "The microscopic section shows a great number of lobules divided transversely, which consist in part of typical miliary tubercles, in part of so-called infiltrated tuberculous tissue, but in part also of what are clearly transverse sections of the remains of glandular acini or sections of perfectly normal acini, pushed widely apart by tuberculous tissue." Giant-cells were found in moderate numbers, tubercle bacilli were sparingly present, "which is the more significant, since the preparation had lain eleven years in Müller's fluid." For this reason, too, the operation of staining was a difficult one. Müller used hand-made sections and "stained them with crystal violet, by Bizzozero's method."

While it is impossible, as Baas says, to suppress a slight doubt with regard to the statement in the latter case as to the method of examining for and the finding of tubercle bacilli, yet it is known, as de Lapersonne has pointed out, that the absence of bacilli in a typically tuberculous tissue cannot be adduced as an argument against the specific nature of the disease.

Baas's own case was that of a teacher, F. W., thirty-two years of age, who had suffered constantly from coryza from his boyhood. In the year previous to his admission a tumor, said to be tuberculous, and which distended and blocked up the nares, was removed from the region of the apertura pyriformis. During the two or three months before admission a hard, circumscribed, nodular tumor, accompanied by injection and swelling of the bulbar conjunctiva and burning pain over the site of the lachrymal gland, had developed. No obvious pulmonary lesion. Urine normal. Other eye normal. The tumor was removed, the wound healed promptly, and the patient disappeared from observation.

Examination of the tumor showed the following characters: In about one fourth of the tumor the glandular structure was still distinctly apparent, but the interacinous connective tissue showed a great increase in its nuclei and infiltration with numerous round cells, so that the acini were pushed apart. In spots the glandular cells had undergone proliferation and become accumulated into masses simulating giant-cells, with multiple, peripherally placed nuclei. Over the remaining three fourths of the tumor, the glandular structure was largely obliterated by the excessive proliferation of the interstitial tissue and by the presence of numerous veritable lymphoid tubercles. None of the tubercles showed typical caseation, but the difficulty with which their centres took up stains proved that a degenerative process had begun there. True giant-cells were found in both the tubercles and interstitial tissue. Mast cells were also present in considerable numbers. No tubercle bacilli were found.

In looking over the microscopical preparations of the clinic, Baas found the records of another case presenting essentially the same histological characters. The tumor here developed in a woman, K. W., aged fifty-nine, in whom the growth had lasted six weeks, and had been attributed to a blow. It had caused no inconvenience except a slight impediment to the movements of the eyeball. Removal was followed by rapid healing without recurrence.

In these cases, in spite of the absence of caseation and of tubercle bacilli, there could be no question about the tuberculous character of the growth. The lachrymal gland behaves in these particulars like other glands which are the seat of a chronic tuberculous hyperplasia.

The presence of numerous dilated and infiltrated lymph-passages make it probable that the infection took place by means of the lymphatic system. It seems likely that the antibacterial properties of the tears have in these cases an influence in delaying the progress and spread of the disease.

Explanation of the Figures.

Fig. 1.—Section of tumor in case of K. W. (Zeiss, Oc. 2., Obj. AA).

a, a_1, a_2, lymphoid-cell tubercles in various stages of development.

b, b_1, dilated, infiltrated lymph-passages.

Fig. 2.—Section of tumor in case of F. W. (Zeiss, Oc. 2., Obj. DD).

a, Epithelioid-cell tubercle with beginning infiltration.

b, Excretory duct of gland.

c, Remains of gland-acini.

Fig. 3.—Small epithelioid-cell tubercle, with a giant-cell, from case of K. W. (homogeneous immersion).

Distinct reticulum ; commencing necrosis in the interior of the giant-cell.

Fig. 1.

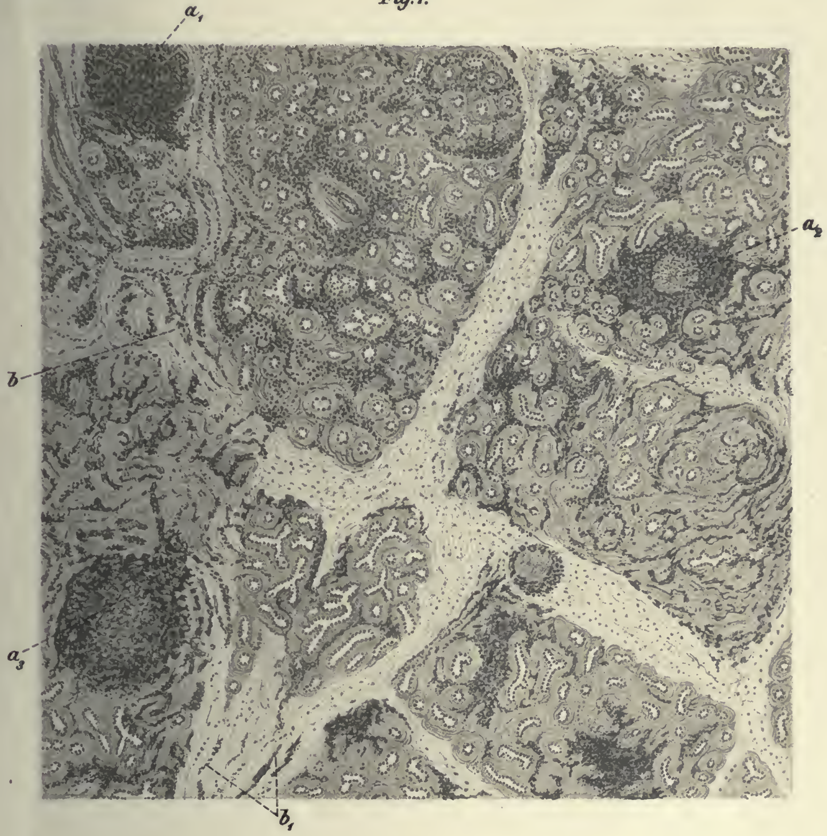


Fig. 2.

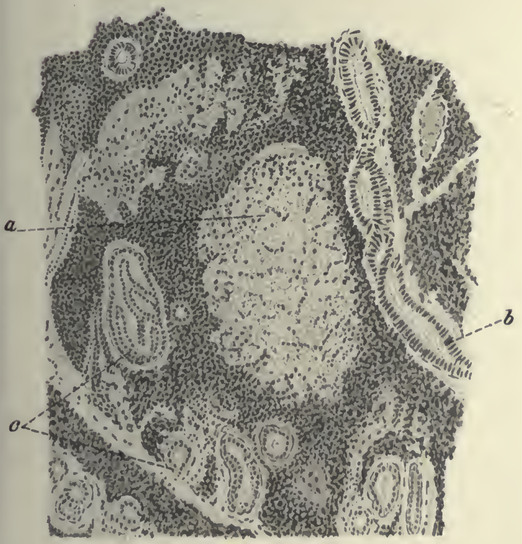
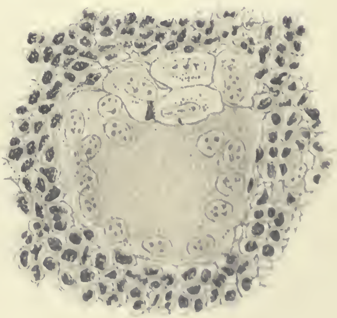


Fig. 3.



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THE CHANGES IN THE CILIARY BODY FOLLOWING PUNCTURE OF THE ANTERIOR CHAMBER. A CONTRIBUTION TO THE STUDY OF THE INTERCHANGE OF FLUIDS IN THE EYE, AND THE FORMATION OF FIBRIN IN THE AQUEOUS HUMOR.

By Dr. RICHARD GREEFF. Privat-docent, Berlin.

Translated (abridged) by Dr. Ward A. Holden.

(*With Plates VII. and VIII., Vol. XXVIII., Germ. Ed.*)

WHEN the anterior chamber of an animal is punctured, the aqueous humor which escapes is clear and fluid and does not coagulate on standing. With the microscope no fibrin is found, and by chemical analysis no albuen; the fluid thus differing considerably from the serum of the blood.

If the anterior chamber is punctured a second time the aqueous humor which escapes is clear but it coagulates immediately. This fluid is rich in albumen and in the coagulated mass is much fibrin.

Some hours later there is usually a deposition of fibrin in the anterior chamber in the form of a delicate membrane, which disappears in a short time. The second fluid differs from the first in containing fibrin, and resembles blood serum in its composition.

Hence we must conclude that from the puncture, the evacuation of the aqueous, and the rapid refilling of the aqueous chamber, there are brought about abnormal conditions which make it possible for substances to pass from the lymph into the aqueous humor that in the normal secretion of aqueous humor are held back.

This idea is supported by an observation of Nicati (*Arch. d'opht.*, ii., p. 25), namely that when a 1 per cent. solution of fluorescin is injected under the skin of a rabbit it does not appear in the aqueous humor, but if the clear aqueous is evacuated, at once a thick green fluid passes from behind the iris and sinks down to the bottom of the anterior chamber, the puncture thus leading to the passage of the fluorescin from the blood into the aqueous humor.

The fact that the composition of the aqueous is not constant has long been recognized, but the cause of the appearance of fibrin after the evacuation of the aqueous has not been explained. For the solution of this and other questions I undertook a series of experiments on rabbits, in the Senckenberg Pathological Institute in Frankfort at the suggestion of Prof. Weigert.

The aqueous humor was evacuated from the eyes of rabbits, and the eyes were enucleated at various intervals thereafter and examined microscopically. Remarkable changes were found, not in the iris or anterior chamber but in the ciliary processes alone.

Immediately after the puncture, numbers of large vesicles appear in the ciliary processes (Plates VII. and VIII.), formed by the elevation of the epithelial covering.

The contents of the vesicles take the stain, and consist of the same coagulated masses that are found in the anterior chamber after puncture, with clusters of red blood corpuscles and occasional white ones.

The vesicles are very numerous, appearing in every section, but they are more numerous in the anterior portion of the ciliary processes where the cells of the pars ciliaris retinæ lose the cylindrical form which they have farther back and become flatter. The cylindrical cells far back are rarely detached, and the wall of the vesicle consists almost always of a flat epithelium with scattered nuclei. This condition is well seen in the eyes of albino rabbits (Plate VII.). In pigmented animals the pigment layer is also detached for the most part (Plate VIII.). The lamina vitrea of the ciliary body is usually detached also and ruptures only at a late stage (Plate VII.). It may remain in position, however, and only the pars ciliaris retinæ and a portion of the pigment epithelium be detached (Plate VIII.).

The sections were stained by van Gieson's method, first deeply with hematoxylin and then with a mixture containing picric acid and acid fuchsin, the connective tissue staining a bright red and the nuclei violet.

The vesicles begin as small flat elevations of the epithelium without coagulated contents, or as cavities in the stroma of the ciliary processes without displacement of the epithelium. The vesicles soon grow large, burst, and evacuate their contents into the posterior chamber. The process of recovery is more difficult to understand. The elevated epithelium might be cast off and the defect filled by proliferation from the sides, or the epithelium might become re-attached after the bursting of the vesicle. Ten minutes after the puncture all stages of vesicle formation may be found in the hardened specimen, small flat elevations of the epithelium, incipient vesicles, numerous large vesicles, and vesicles collapsed. Twelve hours after the puncture small developing vesicles may be found, and even after three or four days a certain number of vesicles are seen, which gradually become fewer, and after six days no traces of vesicles remain.

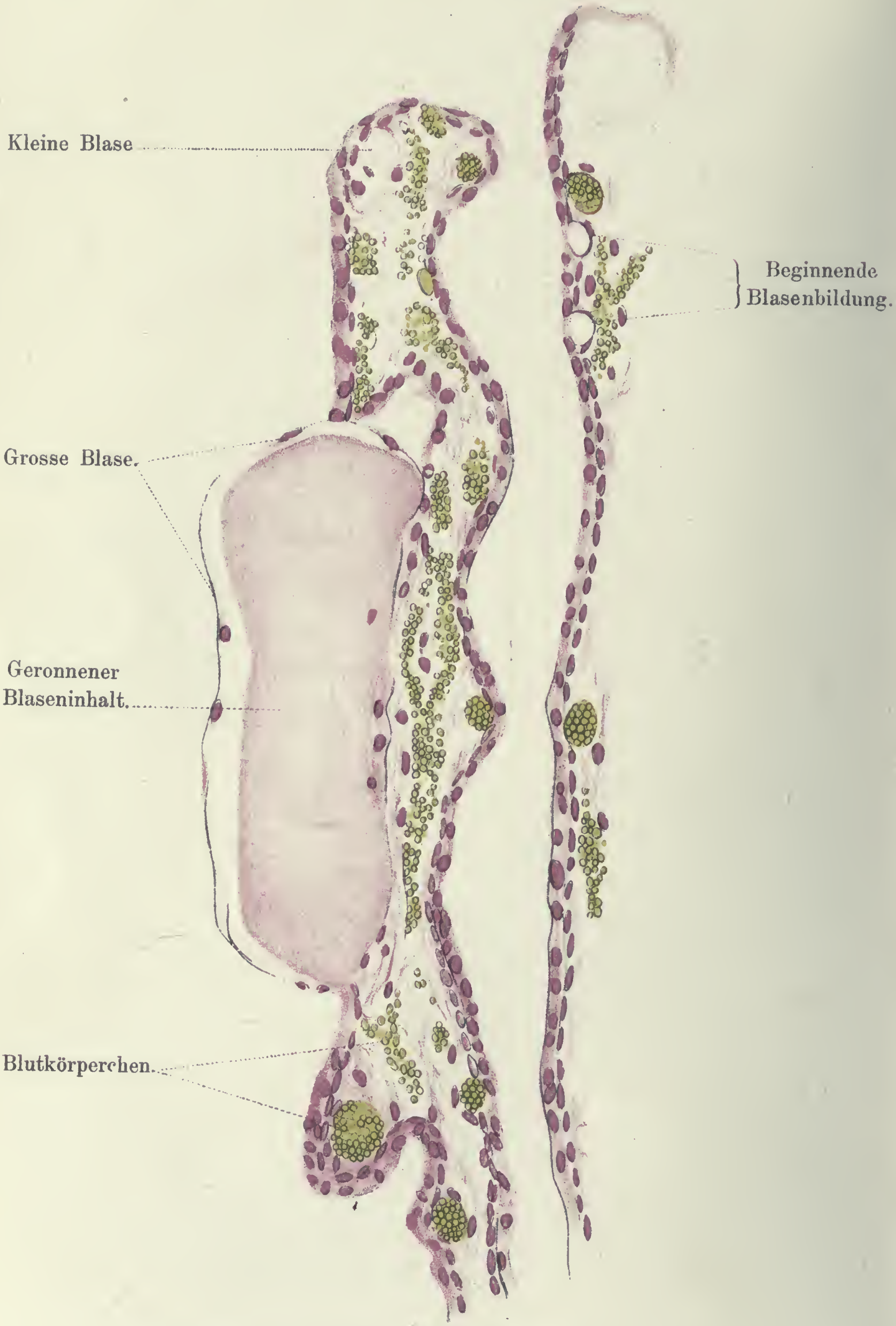
Besides the vesicle formation there is a noticeable hyperæmia of the ciliary processes and a marked œdema with some small hemorrhages.

The conditions found here indicate the seat of the secretion of the aqueous humor. Leber in 1873 considered the ciliary processes the secreting organ. Later, injections of coloring matters into the blood according to Ehrlich's method gave many experimenters varying and unreliable results on account of the diffusion of the coloring matters. More recently Nicati, in his well known papers, and Treacher Collins have confirmed Leber's original views.

We finally come to the question how the sudden appearance of fibrin in the second aqueous humor is to be explained. Nicati is the only writer that has given this subject particular attention. After making a variety of experiments he concluded that the fibrinous secretion was a reflex phenomenon following the decreased intraocular tension. This view does not harmonize with what we are taught by general pathology in regard to the origin of fibrin.

In 1877 Professor Weigert made the statement that fibrin forms only where a spot, beneath which there is a capillary layer, is deprived of its epithelium ; *a priori* then we should expect to find fibrin in the anterior chamber only when there had been changes in the epithelium at some point, and with this idea my experiments were undertaken. Changes in the epithelium were first sought for in the anterior chamber, but were found as described above only in the ciliary processes. As soon as the epithelium here is elevated by the excessive secretion, the active part which it plays in the process of secretion ceases, and albuminous substances which are normally retained now pass out of the serum and form fibrin.

Fig. 1.

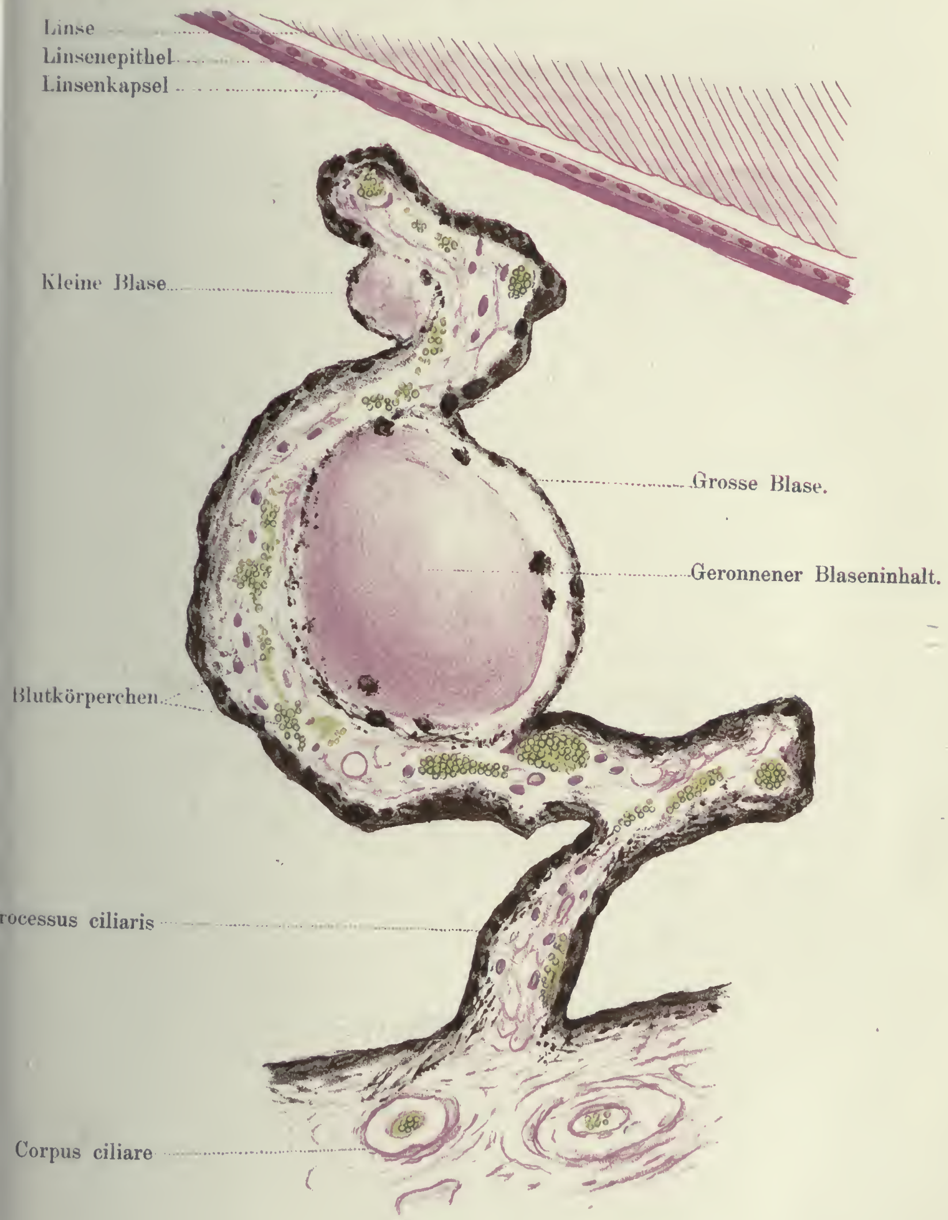


Blasenbildung an dem Processus ciliares nach Punction der vorderen Kammer.

(Albinotisches Kaninchen.)

VERLAG VON J. F. BERGMANN, WIESBADEN. KGL. UNIVERSITÄTSDRUCKEREI VON H. STÜRTZ, WÜRZBURG.

Fig. 2.



Blasenbildung an dem Processus ciliares nach Punction der vorderen Kammer.

(Pigmentreiches Kaninchen.)

Ad nat. delin. Dr. R. Greeff.

VERLAG VON J. F. BERGMANN, WIESBADEN. KGL. UNIVERSITÄTSDRUCKEREI VON H. STÜRTZ, WÜRZBURG.

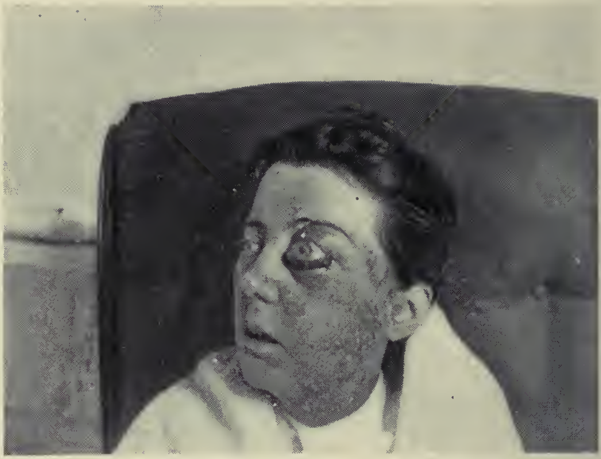


FIG 1.

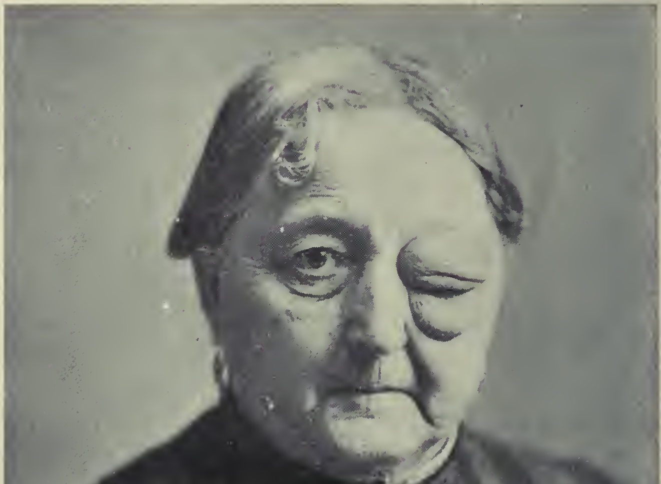


FIG 2.

CLINICAL CONTRIBUTIONS.

By Dr. J. A. SPALDING, Portland, Me.

(*With a plate in the text.*)

I.—EMPYEMA OF THE ORBIT DUE TO EXTENSION OF DISEASE FROM A NECROSIS OF THE ALVEOLAR PROCESSES OF THE SUPERIOR MAXILLA ORIGINALLY EXCITED BY EXPOSURE TO PHOSPHORUS FUMES IN A MATCH FACTORY.

(*With Fig. 1. of the accompanying plate.*)

EARLY in March, 1893, I was asked to look at a patient in the Maine General Hospital who was said to be suffering from severe pain in the left eye.

The history of the patient was as follows:

Early in the autumn of 1892, after working for some months steadily in the match factory in Portland, she first perceived a roughening of the gums, first in the upper and then in the lower jaw, and progressing rapidly. Soon after some of the teeth felt rather loose and began to decay very fast. Next, the bone beneath the teeth seemed soft, and finally the alveolar process on the left side of the superior maxilla began to crumble, and the region above to be painful nearly all the time. As no improvement followed attempts at local treatment, the patient consented to an operation in the winter, at which five teeth were removed, and then from between the plates of the superior maxilla a large sequestrum nearly one and three-quarters of an inch long was removed. The surgeon who had charge of the case, Dr. Weeks, and his assistant, Dr. Brock, found no other cause for the maxillary necrosis than the exposure to phosphorus fumes. This is, however, so common an occurrence amongst a certain class of employés that it is a legitimate conclusion to draw, that phosphorus causes maxillary necrosis under favorable conditions, the chief being when the teeth are decayed. Whether employés with good teeth have no phosphorus necrosis I am unable to state, but this is a point which might profitably be studied by those who have opportunities in this direction. At the same time attention could be given to the question whether good teeth do not longer fortify the alveolar process from necrosis.

After her recovery from the effects of the operation, the patient felt so much relieved that she resumed her work for a short time, but new foci of the necrosis making their appearance, she left the factory and kept at home. In point of actual fact, however, she had no idea of asking for advice or treatment of the renewed pain higher up, in the jaw, and even behind the eyeball, or even for the rapid protrusion of the globe which began in February. For it was not until Dr. Brock accidentally met the patient with her face and eye bandaged, and uncovered the condition of affairs as exhibited in the subjoined photograph (Fig. 1.), that at his urgent appeals she consented to enter the hospital for an examination at the least, if not for a capital operation.

On removing the bandage which had been a necessity for several weeks, the appearance as exhibited in the annexed photograph met my sight. The photograph requires to be looked at very carefully in order to bring out all the points in this extraordinary case, for the light not being so good in the ward as one would desire, the difficulties of making a good picture were many. Still we get a reasonably good idea of the aspect of the parts, though the *prominence of the left eyeball is not enough pronounced.* In fact, I think that the patient had to be depicted in bed, owing to difficulties in moving her on account of the pain in face and jaw. It seems to be on the safe side of exaggeration to state that the left eye projected so far in front of the other as that its posterior aspect was on a level with the cornea of the right eye. It had "*laid out on her cheek*," as the patient expressed herself, for a few days, advancing at first rapidly with violent pain, and then slowly with less pain. The patient lacking intelligence, it was hard to discover the actual amount of vision before the protrusion of the globe, as well as the existence of any diplopia. At all events she asserted that there was at first double vision, then loss of what sight she had, so that at her admission the eye was totally blind. Even if the pressure on the optic nerve had not destroyed sight, the rapid desiccation of the cornea from constant exposure to the atmosphere, day and night, would soon have abolished whatever vision might have been left. Nothing could be done with the ophthalmoscope owing to the desiccating of the cornea. Still it is very curious to observe in the photograph the brilliancy with which the cornea, iris, and pupil are defined, despite the fact that to the naked eye all these structures were so much obscured that it was difficult to see them at all. This extraordinary definition of these parts by the photograph may be analogous to the photographic visibility of skin eruptions, *e. g.*, previous to small-pox, etc., long before the eye can see a trace of the same.

The eyeball could not voluntarily be moved in any direction. The upper lid was very tense and extremely sensitive to the touch. The lower lid was more œdematous than the upper, but not sensitive. Pressure on the globe backward excited violent pain.

The question now arose concerning the best means of procedure. There was evidently before us a case of empyema of the orbit. The necrosis of the alveolar processes, excited by phosphorus fumes, had extended to Highmore's antrum, and from there pus had made its way into the orbit. It did not seem possible to evacuate the pus from the orbit by incision through the conjunctiva below the globe, for if there were extensive orbital disease it would be most difficult to heal by daily syringing of any remedies through the conjunctival orifice of the incision. And if the orbit were necrosed, proper scraping could not possibly be done through a small incision requiring constant attention to prevent early closure. We might have opened the antrum from below by the classic operation, but the patient was averse to the repetition of an operation similar to the first that had been performed with so little benefit to her malady. Then, too, the cornea was in a state of incipient necrosis, so that, triumphant as it would have been to save the eye, and cure the progress of the disease, it did not seem possible of accomplishment. For all these reasons enucleation was decided upon, then the orbit was to be cleansed to decide its condition, and indications were to be followed as they might present themselves in the near future.

The **operation** was performed under strict aseptic precautions as soon as a decision could be reached, and after removing the globe an enormous amount of profoundly offensive pus rolled slowly out of the orbit. The floor of the orbit was found largely necrotic, and large probes and spoons could be easily passed into the antrum. In point of fact, the bone was so much diseased and friable that the boundaries of the antrum could not be defined at all. Whatever the sharp spoon could do was done effectually, then the orbit was packed with iodoform gauze, and from that time onward daily cleansed with antiseptics, and packed with gauze.

The eyeball was intact so far as concerns the shape, and there was no noticeable macroscopic lesion of the interior. The optic nerve, a piece of which, nearly 2 *cm* in length, was removed together with the globe, was pressed into a thin ribband-shaped structure, and upon microscopic examination proved to be totally atrophic from pressure.

The patient made a rapid recovery from the orbital affection at first, but soon the pus was again secreted in abundance, and fresh necrosis of the maxilla demanded the surgical removal of the entire bone, which was done about two months after the enucleation of the globe. For a time all went well, but in the autumn the patient was suddenly attacked with facial paralysis on the left side, later still with left hemiplegia and aphasia, and at length died from exhaustion and paralysis.

Cases of this nature are very rare, for, though phosphorus necrosis of the jaw is not uncommon in match factory employés, the extension of the disease through the antrum and into the orbit is very rarely reported. The employés generally run from the danger ere it is too late, but our patient took too little care of herself after the disease had begun, to say nothing of returning to work with an operated and unhealed jaw subsequent to the removal of the sequestrum.

If I had another similar case I should not hesitate to urge the excision of the superior maxilla first, as the preliminary to proper treatment of the orbit. I feel sure that if we had urged this step here we could have evacuated the pus and scraped away the necrosed bone, and with constant cleansing delayed the fatal issue much longer, even when the state of affairs was so bad as at the original entrance of the patient into the hospital.

To those who are interested in cases of this sort I will say that similar ones are mentioned in the *Archives of Otology*, vol. xxii., p. 312, in an instructive article by Dr. H. Knapp, the editor, whilst in another part of this number I have printed still another case bearing on the occasional connection of diseases of the antrum and orbit through the sphenoid or ethmoid cells.

II.—A CASE OF UNILATERAL ŒDEMA OF BOTH EYE-LIDS WITH EXOPHTHALMOS AND PARTIAL OPTIC ATROPHY PERSISTING UNALTERED FOR THIRTY YEARS.

(*With Fig. 2 of the accompanying plate.*)

IN connection with and following on the case of exophthalmos from empyema of the orbit excited by extension of mepyema of Highmore's antrum after phosphorus necrosis (published in the present number of these ARCHIVES), I beg leave to offer a *rara avis*, an absolutely unique case in ophthalmology, so far, at least, as I am able to discover in a rather extensive mass of literature at command.

After first of all calling attention to the subjoined photograph (Fig. 2), in order to get a clearer idea of the condition of affairs than any words can give, I will make a brief abstract of the patient's own account of her very interesting and extraordinary condition.

"The first I felt, more than thirty years ago, that there was anything the matter with my left eye, was that the upper eyelid began to droop, at first very slightly, and then in the course of months or possibly a year or two, more and more perceptibly, till it closed down over the eyeball and the sight left me."

Let me here interrupt the patient by saying that the expression "the sight left me," is to be taken with circumspection. It simply means that, the eyelid drooping so far down over the cornea, the sight was abolished so long as the lid was not actually lifted up to uncover the cornea. For, as we shall see farther on, at the time when I first tested the sight it was not inconsiderable even after thirty years of asserted loss. Many a *blind* person recovers excellent sight when the inflammatory swelling of the eyelids is reduced so that light can enter the uncovered pupil. How soon the sight in this case was actually lost after the beginning of the œdema of the eyelids is, therefore, a question largely of surmise.

To return to the patient's narrative:

> "I lost my sight as near as I can remember twenty-five years ago, but I had no idea that the eye was lost till the sight was actually gone from it. The eye that was swelling was drawn, and that saved the other eye from going the same way, or at least the physician said so." (By way of parenthesis, let me say that to "draw" an eye, in New England *parlance*, means to raise a large blister on the temple or behind the ear on the same side with the eye affected.) "The upper lid first swelled, as I say, and after some months the eye began to bulge very slowly forward. Then the sight seemed affected, and after a long time the lower lid began to swell in the same fashion as the other had done. After the lids became once swollen they remained in the same condition for many years, only I have often noticed that if I have a cold on my lungs or in my throat and have a blister drawn on my back or chest for relief, then the swelling of the eyelids decreases noticeably, and I can lift the eyelids apart with greater ease. At those same times it seems to me that the sight is a great deal better, for I can by lifting the lids see to count the window-sashes across the room, and even to see large type. At other times, even with the lid held up, it seems as if there were no sight in the eye except enough to see a lamp burning in the room.
>
> "I suffer no pain now, in or about the eye, have no headache, and if it were not for the heaviness about the eyelids I should never think of the trouble at all except when I see my face in the looking-glass. My health leaves nothing to be desired in any respect."

I cannot add much to the description of the external appearances of the patient that the photograph does not tell. The tissues about the swollen lids are not, strictly speaking, of an œdematous feeling. They are very soft to the touch, whilst œdema feels hard. I should describe the sensation as like passing one's hand over velvet or plush. On raising the upper lid, which yields easily to the finger, the globe comes into sight, protruding about ½ of one *cm* in front of the level of the other eye. It can be rotated freely in every direction, except upward and outward, where it seems to meet with some obstruction which however cannot be felt through the lid. Whether the obstruction is due to a tumor or not is difficult to define. Possibly the mere swelling of the lids suffices to act as an obstacle. The lower lid is also swollen and is rather harder to the touch than the upper. There is no pain on pressing against the globe through the lids in an effort to push it back into the orbit. On pressing both lids apart the conjunctiva of the globe and lids seems rather tense, slightly chemotic and congested, but not very abundantly so. The cornea is normally transparent, iris normal in color with the other, and the pupil though rather wider than that of the right eye reacts promptly to light and shade, and also consensually with the other, which is a good sign of existing vision. The ophthalmoscope shows the media transparent, and nothing abnormal but an *exceeding whiteness of the optic disc*, from which one would immediately diagnosticate optic atrophy were not the remaining sight reasonably good. Snellen 20 at one foot to 15 inches. Visual field normal. The other eye is normal and the patient uses a moderately strong convex lens for near vision.

There are no bodily abnormities, and otherwise than the deformity from the left eye and lids, the patient is free from disease.

The patient declining any interference even in the way of an exploratory incision into the orbit, the ætiology of these remarkable appearances must remain in doubt. Œdema of the eyelids is almost always due to some constitutional disturbance, preferably in the heart or kidneys. But here there is nothing abnormal in either of these organs, as careful examination and urinary analysis revealed. We are therefore presumably led to the conclusion that there is some stasis in the venous circulation in the orbit, or some mucocele or empyema in the ethmoid or sphenoid cells which retards the circulation.

Œdema of the eyelids being a symptom around the eyes but not a disease of the latter all treatment is purely empirical, and must be based on such constitutional conditions as we can discover. Here nothing being wrong we can only wait and watch.

ON OCULAR AFFECTIONS IN SYPHILIS OF THE BRAIN, WITH REPORT OF FIVE CASES.[1]

By CHARLES ZIMMERMANN, M.D., Milwaukee, Wis.

IN two previous papers which I had the honor to present to this society (*Transactions Wis. State Med. Soc.*, 1891 and 1892, and *The Medical and Surgical Reporter*, July 29, 1893, and Nov. 25, 1893) I endeavored to describe the intimate connection between the eye and the brain. The optic path, the third, fourth, and sixth nerves run the greatest part of their length within the cranial cavity, so that they become largely exposed to encephalic diseases. Not only the variety and frequency of functional disturbances, owing to this arrangement, but also the possibility of inspecting, with the ophthalmoscope, the entrance of the optic nerve in the eye and the retina, give a special value to the eye symptoms in affections of the brain. Therefore it will not be surprising that in syphilis, which may invade the brain in all its structures in various fashions, only about 15 per cent. of the cases are without ocular symptoms (Uhthoff).

The literature of this field is abundant, but only in the last years, with the perfection of methods and the great progress in examining the brain and nerves anatomically and especially histologically, have the observations become more accurate. One of the latest works is Uhthoff's admirable paper, based on 100 cases of brain-syphilis with ocular symptoms, observed by himself, of which 17 came to a post-mortem examination, and on 150 autopsies, compiled from literature (*von Graefe's Archives*, vol. xxxix., 1 and

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[1] Read at the Wisconsin State Medical Society, May 2, 1894.

2, and vol. xl., 1). In the following I purpose to speak of ocular affections in syphilis of the brain, with the report of five cases of my own observation. The examination in all these has been very careful, but for the sake of brevity I shall omit the negative results, and state the positive ones only.

CASE 1.—*Hemiplegia, hemianopia, etc., from basal gummous meningitis.*—A man of sixty-two years was paralyzed in June, 1891. Syphilitic infection seven years previously. September, 1891, I saw him for the first time in consultation on account of impairment of sight, and ascertained right hemiplegia (face, arm, leg) and right homonymous hemianopia. V. $\frac{15}{20}$. Ophth. normal. Tongue deviates to the right when protruded, to the left when drawn back. Defective articulation, speech often unintelligible, failure of memory, amnesic aphasia. The right lateral hemianopia sometimes was complete, sometimes there was only hemiachromatopia, *i. e.*, all appeared gray in the right half-field, or hemiamblypia, *i. e.*, objects could be seen, but dimly only, and seemed to be smaller. For instance, when looking at me with either eye, the left side of my face, my left eye, and hand appeared to him smaller, as if they belonged to a smaller person. Antisphilitic treatment without result. The condition grew gradually worse. Dec. 29th the patient was lying on the lounge perfectly apathetic ; did not speak ; incontinence of urine and fæces. The ophthalmoscope revealed in both eyes very marked papillitis with slight hemorrhages into the substance of the optic discs. Tortuous engorged veins. Outlines of discs veiled, which were rather swollen and prominent. Pupils medium-sized, react very sluggishly to light. Jan. 1, 1892 : Pulse, 120. Excessive perspiration. Very restless. Right limbs painful when he is dressed. Jan. 6th, death, 9 P.M.

Jan. 7th, 1 P.M., I made the *autopsy:* The base of the left hemisphere is the seat of a diffuse growth, starting from the meninges, yellowish-gray, of lardaceous consistency, involving the frontal and temporal lobes, optic tracts, pons, middle and posterior cerebral arteries, and the third nerve. This growth is the result of thickening of the pia-arachnoid, due to syphilitic meningitis. It extends into the fissures and recesses and has grown into the walls of the vessels, conglomerating all these different portions of the brain into one mass. The trunk of the middle cerebral artery is dilated, and its coats are invaded by the gummous growth which sheathes the artery all around. Its branches lead to a large focus of softening in the corpus striatum from arterial occlusion. Microscopically the growth at the base consists partly of fibrous tissue, and its portion at the frontal lobe is in a state of fatty degeneration, showing granule corpuscles, fatty granules, and detritus.

The features of interest in this case are the homonymous hemianopia and the papillitis. The former was caused by the affection of the left optic tract, which, with the frontal and temporal lobes, was the seat of the syphilitic new-formation, *i. e.*, it was due to a *basal* lesion. Indeed this basal gummous meningitis is the most common condition in syphilis of the brain. It may be diffuse or circumscribed, may be complicated with a tumor-like formation of gummous tissue, or a gumma proper may be the only affection. Or its seat may be the convexity of the brain.

Next in frequency is the syphilitic endarteritis (Heubner), and this may accompany the former. The softening of the brain at the corpus striatum was brought about by compression of the middle cerebral artery by the gummous neoplasm at the base, leading to thrombosis of its terminal twigs, and subsequent necrobiosis of the brain area supplied by it.

The fibres of the visual path are implicated very frequently(in fourteen out of Uhthoff's seventeen autopsies), but the optic tract, as in our case, rather exceptionally, possibly on account of its situation not being so much exposed. The seat of predilection and the starting-point of the gummous affections of the cranial base is undoubtedly the chiasm, which may be entirely imbedded in a gummous mass. This may extend downwards to the optic nerves, leading to a gummous degeneration with considerable swelling, or to optic neuritis or papillitis and perineuritis, with consequent neuritic atrophy or to descending atrophy. Quite a number of post-mortem examinations showed this condition, or gummous masses sheathing the optic nerves, not entering into their substance, without functional or ophthalmoscopic changes during life. Thus we cannot exclude its existence even in cases without corresponding clinical symptoms, if the diagnosis of cerebral syphilis is established by other signs. Mostly it is not a primary gummous process in the optic nerve, but an interstitial affection invading the nerve from its periphery along the septa and replacing its fibres. The *intracerebral* optic fibres are much more rarely damaged than the basal (Uhthoff, xxxix., 1, p. 121).

The visual path may, however, be affected by way of the arteries supplying them. Endarteritis obliterans of the ophthalmic artery is of very rare occurrence (Uhthoff), and in case of obstruction its many anastomoses may prevent blindness by re-establishing the blood supply. Whenever it was observed, the functional disturbances could more easily be explained by direct gummous degeneration of the optic nerves. But endarteritis of the cerebral arteries proper is very frequent. Our second case shows the course of this affection.

CASE 2.—*Right hemiplegia, hemianæsthesia, etc. from syphilitic obliterating endarteritis of the left middle cerebral.*—A man, æt. forty-one, acquired syphilis 1887. In January, 1890, he suffered from severe headache day and night for weeks. He was treated at the hospital for one week, and rested at home for two weeks. The headache continued, however, more or less. July 15, 1890, his physician sent him to me for a laryngoscopic examination, as he complained of severe pain at the left side of the throat in the region of the fifth vertebra. Larynx healthy, but a very large and deep syphilitic ulcer at naso-pharynx. He looked careworn and emaciated ; had lost eight pounds within a few weeks. I ordered large doses of iodide of potassium and treated the ulcer successfully.

Aug. 19th he was led into my office, having met with a paralytic stroke on the way, resulting in right hemiplegia (face, arm, leg). He said that the night before he suddenly felt numb in right side of body, with formication in his right limbs. But he got over it and slept well. This morning he went to his store perfectly well, and when he came to me he met with this accident. Sensibility lost on right side of face and body. He can raise his right arm only slowly and with great effort, but it soon falls down. His right leg is dragged behind and he cannot stand on it. Speech hesitating and in some words not clear. After sitting down and resting awhile he can walk and move his arm better, but he is weak. Sensibility returns. At once sent to hospital and most energetic antisyphilitic treatment instituted. Aug. 27, 1890, walks pretty well and is improving rapidly. After three weeks at hospital he recovered so far that he was able to work as a clerk. I last saw him on April 7, 1894; no relapse in these four years. His weight increased from 121 pounds to 153. Except occasional paræsthesia in right leg and arm he feels perfectly well.

Diagnosis : Syphilitic endarteritis obliterans of left middle cerebral artery, narrowing its lumen and impeding and arresting the circulation. Fortunately only an ischæmia took place without thrombosis, the blood current being strong enough to penetrate the narrowed vessel, preventing thrombosis and necrobiosis. Thus we had only a transient interference with the function. The energetic antisyphilitic treatment commenced at once after this attack arrested the disease most effectually.

According to Heubner (*Die luet. Erkrank. der Hirnarterien*, Leipzig, 1874, p. 186), the white matter of the cerebral base, even the chiasm, the cranial nerves, pons, and medulla are directly supplied by terminal branches of the large vessels in the same fashion as the central ganglia. Thrombosis in these branches from endarteritis obliterans must damage the area supplied by them. Marchand (*v. Graefe's Arch.*, xxviii., 2, p. 86) observed homonymous hemianopia from an infarction in one optic tract, and a case of Treitel and Baumgarten (*Virch. Arch.*, cxi., p. 251, 1888) is analogous. Treitel finds in this condition the best explanation of isolated or partial paralysis of cranial nerves. It is not necessary to find an atrophy in such instances, as the obstruction of the vessels need not be complete, so that only the function, not the nutrition, is abolished.

During life our first case exhibited two symptoms of affection of the visual path: homonymous hemianopia and papillitis. One of these, or optic neuritis, atrophy of the disc, temporal hemianopia, or other disturbances of the visual field was observed in more than 50% of cases of cerebral lues. Papillitis and optic neuritis differ in this: a prominence of the optic disc of at least 2 D. (*i. e.*, 2/3 *mm*) is called papillitis, of less, optic neuritis. Both forms and simple atrophy are about equally distributed (about 12 %). Papillitis is almost always double and, as in our case, most frequently caused by basal meningitis or gummata within the cranial cavity. In some instances it was the consequence of cerebral softening from syphilitic arteritis or of syphilitic periostitis of the orbit. Sometimes it makes the impression of a primary affection, when other cerebral symptoms are wanting, despite the actual existence of intracranial affections. It may disappear, without leaving traces, or may pass into atrophy, but the sight may recover. In our case it developed rather late in the series of cerebral symptoms and made the prognosis worse, as, in cerebral tumors, for instance, its occurrence precedes death by no long interval, if it sets in long after the symptoms of tumor.

Optic neuritis is also most frequent in basal gummous meningitis. Although it is to be considered as a descending neuritis, the intensity of its ophthalmoscopic appearance does not necessarily correspond to the intensity of anatomical changes in the optic nerve, especially when the cause of the visual disturbance lies farther back on the cranial base, as pointed out by Uhthoff. Thus there may be considerable failure of sight, ere slight changes of the disc are noticeable. According to Blessig (*Kl. M. f. A.*, 1875) it often is characterized (in comparison to papillitis) by rapid impairment of vision, less swelling, early grayish-white discoloration, and it may be one-sided. Sometimes stellate small white patches at the region of the macula are seen simultaneously. Primary syphilitic optic neuritis without other cerebral or ocular symptoms is rare. Atrophy of the optic disc, without preceding optic neuritis or papillitis, was observed in about 14 %, and it could always be proven to be secondary, quite differing from the primary gray atrophy of the optic nerve, *e. g.*, in tabes, by the peculiarity of the visual disturbances, especially the visual fields (Uhthoff, xxxix., 3, p. 157). It is mostly bilateral, complete or incomplete or partial, limited to the temporal half, as seen so often in amblyopia due to intoxication from tobacco and alcohol. Its aspect, however, has nothing characteristic of syphilis. Ophthalmoscopic changes of the retinal arteries of syphilitic origin, especially endarteritis, have been considered by Seggel, Ostwalt and others as premonitory symptoms of syphilis of the brain, others (Uhthoff) hardly ever observed them in their cases of cerebral lues and doubt their intimate connection with the latter.

The functional disturbances in syphilitic affections of the visual path within the cranial cavity refer mostly to the field of vision, blindness being extremely rare. Homonymous hemianopia, as seen in two of our cases, is quite common and often of basal origin (in 4 out of Uhthoff's 11 cases). If it is basal, as in our first case, it is due to an affection of one optic tract, and this is much more common in syphilis than in any other brain disease. It is marked by hemianopic reflex immobility of the pupils, by simultaneous affections of other cranial nerves, or by subsequent hemianopia of one or both remaining half-fields, when the chiasm becomes invaded, and by positive ophthalmoscopic changes of the optic discs, after a certain time, which generally consist in pallor. We had bilateral papillitis, seen four months after the first observation of hemianopia. It may have set in earlier, but I had not seen the patient for over a month. If he had lived longer, the papillitis might have turned into atrophy. According to literature temporal hemianopia occurs about twice as often (20 %) as homonymous (10 %) in syphilis of the brain, and is due to affection of the chiasm. If the visual disturbance is at first limited to one eye and temporal hemianopia sets in, the disease started in the optic nerve and spread to the chiasm. When commencing with homonymous hemianopia it originated in the optic tract and extended to the chiasm. In either case the non-decussating fibres of the other eye will be spared to the last, and before these are affected, the temporal hemianopia is one-sided. Later on there will be always ophthalmoscopic changes of the optic discs, optic neuritis or atrophy. Other forms of disturbances of the visual fields, as concentric limitations, central scotomata, enlargement of the blind spot or irregular defects with preservation of a peripheral crescent are occasionally observed and are mostly due to syphilitic disease of the optic nerve-trunks, generally associated with papillitis, ophthalmoscopically visible, but at first the latter may be wanting.

Total amaurosis in both eyes is very rare, and when observed it was mostly transient, yielding to antisyphilitic treatment.

This fact, as well as the form of the disturbance of the visual field, the oscillating course, and the complication with paralysis of other cranial nerves or parts of the brain are characteristic of the ocular symptoms in cerebral syphilis. Oppenheim explained this wavering of the visual complaints by the anatomical character of the syphilitic tissue: "This rapidly growing granulation-tissue exuberates and as rapidly decays in constant repetition, exposing the nerve invaded by it to ever-changing pressure."

The next frequent eye affection in syphilis of the brain is that of the ocular nerves. In Uhthoff's collection, out of 167 autopsies (150 + 17) the optic fibres were diseased 85 + 16 = 101 times, the third nerves 56 + 10 = 66, the sixth 26 + 3 = 29, the fourth 5 + 1 = 6, the fifth 22 + 3 = 25 times. Lesion of the third nerve, or both, is almost always basal, and is due to a gummous meningitis of the interpeduncular space or to gummata in this neighborhood. The gummous products not only act by pressure, producing atrophy, but infiltrate the nerve sheath and the nerve itself, leading to gummous perineuritis and neuritis. Or they develop more directly in the nerve, which degenerates, setting up syphilitic neuritis of the roots (Kahler), with swelling and proliferation of blood-vessels, or converting the nerve completely into gummous formations. Paralysis of one third nerve with crossed hemiplegia is due to gummata or foci of softening in the region of the peduncles or the pons, in consequence of thrombosis from syphilitic arteritis or from compression of the vessels at the base by the products of gummous meningitis.

This was observed in our *third case :*

A man, æt. thirty-two, had a primary sore 1891. March, 1893 paralytic seizure in bed (while travelling). A week later I saw him in consultation with his physician at his residence ; *crossed hemiplegia, affecting limbs on right side, face on left, with total paralysis of left third nerve.* Ptosis, mydriasis, immobility of pupil, crossed diplopia, inability to read. V. in each = $\frac{15}{30}$. No ophthalmoscopie changes. Tongue deviates to the right when put out; articulation slow and indistinct. Memory weak, has to think about words before he can utter them. He can walk, but very slowly. Antisyphilitic treatment without much result. I did not see him again until July, 1893, when he walked to my office: About the same condition. Ptosis and accommodation have improved. He reads No. 1, with either eye in 20 *cm*; pupil more than medium wide, contracts a little to light. April, 1894, ptosis entirely gone. Divergent squint; the left eye can be moved to the middle line, but not beyond. Pupil, 6.50 *mm* wide (R. 2.50 *mm*), reacts a little to light. He is not annoyed by double images, which he ignores. Psychically he is worse; he is not able to write a letter, forgetting what he wants to say. His left arm and leg tremble, and he can write his name tremulously only with great effort.

Diagnosis Syphilitic affection of left side of pons.

When both third nerves are paralyzed from syphilitic new-formations in the interpeduncular space, it may happen that the most medial fibres are chiefly damaged. Although the posterior cerebral artery winds around the third nerve, it has not been found by Uhthoff in any of his cases causing oculomotor paralysis by compression or by thrombosis of its twigs supplying the nerve. From the fact that the affection of the third nerve in syphilis of the brain is mostly basal, we would infer that the paralysis observed during life would be total, according to the law that a partial ophthalmoplegia is either nuclear or fascicular, *i. e.*, where the nuclei and fibres of the different branches are separated. But anatomical researches have shown that apparently complete affections of the trunk of the third nerve at the base have caused only partial paralysis, viz., either ophthalmoplegia exterior or interior, isolated ptosis or paresis of only one or a few of the muscles supplied by the third nerve, or no symptoms at all as in our first case.

Our *fourth case* shows such a *partial paralysis of the third nerve of basal origin.*

A man, æt. thirty-seven, had a chancre thirteen years ago, and was treated for several years. Nine years ago he consulted some authority in Europe, who pronounced him cured, and consented to his marriage. He, his wife, and children did not show any trace of syphilis during that time. Since December, 1893, he suffered from severe headache, but did not seek any medical aid. February, 1894, he suddenly felt sick, and was paralyzed (without losing consciousness), so that he could not walk any more. A week later I saw him, in consultation, on account of his eyes. Right hemiplegia, right ptosis, paresis of right internal rectus, and myosis. Right pupil 1 *mm* smaller than left. Crossed diplopia, inability to read with both eyes. Singultus since the attack lasting day and night for a week, disturbing his sleep and meals. Retention of urine in the first week. After six weeks the ptosis subsided, but returns a little towards evening, or when he is tired. No more double images. He goes out since that time, but his gait is staggering and uncertain. He complains of severe headache, mostly in occiput, but his mental condition is not much changed. Since April 1st his left arm has become weaker, which from the onset showed changes in the sense of temperature (all objects feeling warmer than the right), and his left leg has become ataxic.

The considerable variations of symptoms, the irregular palsies of limbs and cranial nerves (third) in this case, and the gradual deepening of the apoplectic seizure suggest a widely spread pathological process at the cerebral base. This may be either extensive basal syphilitic meningitis, injuring indirectly the vessels on the left, the third nerve on the right hemisphere, or syphilitic disease of the basilar artery with occlusion or incomplete obstruction of arteries that arise from it. The myosis may be due to an irritation of the pupillary fibres by the same process which paralyzed the other branches of the third nerve (analogous to Fontan's case, quoted by Mauthner, but explained as a nuclear affection).

In some cases of isolated ptosis the microscopical examination proved the nuclei to be healthy, but the nerve trunk diseased; a cortical lesion could never be found as cause (Uhthoff xl., 1, pp. 63 and 67). The paralysis of the intrinsic or extrinsic muscles produced by a basal affection must be considered as an exception to the rule, which is, that it is nuclear or fascicular, but it may happen occasionally. Fascicular palsy of the third nerve was noticed in connection with crossed hemiplegia due to syphilitic disease in the pons or crus.

Paralysis of the sixth nerve, one or both, is in most instances basal from gummous meningitis, and mostly conjoined with palsies of other cranial nerves, or from endarteritis of the basilar artery with its sequelæ. If it is intracerebral, the lesion is in the pons. In none of the sixteen cases mentioned by Uhthoff could a nuclear affection be detected.

Our *fifth case* may illustrate this condition.

A man aged thirty-four had primary syphilitic infection, 1884. I saw him, in consultation, September, 1891. He had had eruption of skin, psoriasis plantarum, sores in mouth, pain in shinbones. For about eight weeks he had headache with sleeplessness, and complained of his eyes. *Paralysis of right abducens.* $V = \frac{15}{15}$ in each, no ophthalmoscopic changes. *Right homonymous partial hemianopia.* The hemianopic field is very irregular. Above and below it nearly reaches the median line (lacking 10 °). In some meridians it extends to the fixing point, but the periphery is partly preserved. This irregular defect is homonymous in both eyes. His headache commenced on the left side, and is now on the right. He is under great psychical depression, and cannot work. His memory of place (topographical ideation) has somewhat suffered, as he several times had difficulty to find his home, being confused about the streets. Antisyphilitic treatment improved his headache, but the paralysis of abducens did not disappear until summer, 1892. At that time his wife was confined with a child, who died with pemphigus after six months, according to the family physician, who also stated that the first child, five years old, is suffering from pemphigus. I did not see the patient any more until April, 1894. He is of healthy appearance and feels well, but has occasionally headache. Paralysis of abducens has entirely disappeared. $V = \frac{15}{15}$. Ophthalmoscopic condition normal, but the right partial homonymous hemianopia is just the same as at first.

Diagnosis: The absence of ophthalmoscopic changes of the discs after two years, the partial homonymous hemianopia, and the defective memory of place (Foerster, *v. Graefe's Arch.*, xxxvi., 1), would rather indicate a lesion of the visual centre in the left occipital lobe, than of the left optic tract, but the paralysis of the right abducens was due to an affection in the right hemisphere. For these widely separated foci the best explanation would be, that a basal gummous meningitis injured on the right side mostly the nerves (abducens in this case), on the left the arteries (posterior cerebral), leading to thrombosis of some of its branches which damaged portions of the occipital lobe supplied by them. Oppenheim (*Berl. klin. W.*, 1889, p. 1036) observed such conditions in hemiplegia with paralysis of cranial nerves of the same side. Our fourth cases with right hemiplegia and partial paralysis of right third nerve, is another instance of this category. Or it may be interpreted by an arteritis of the basilar artery, predominating in the left posterior cerebral and the right anterior inferior cerebellar branch. The analogy with Marchand's and Treitel's cases, mentioned above, would admit of a third explanation, viz.: The arterial disease may have settled in a terminal twig supplying the left optic tract, with consequent partial functional damage to the tract without following ophthalmoscopic changes. In that event, however, we might have expected a subsidence of the hemianopia as well, as the paralysis of the abducens ceased, the nerve fibres being more resistent than the brain substance of the centres. Thus, besides the other points mentioned, the recovery of the abducens, but the persistence of hemianopia, would be in favor of an affection of the occipital lobe and not of the optic tract.

PERMANENT CENTRAL SCOTOMA CAUSED BY LOOKING AT THE SUN DURING AN ECLIPSE, AND COMPLICATED BY UNIOCULAR, TRANSIENT, REVOLVING HEMIANOPSIA.

From Dr. Knapp's Practice, Reported by

Dr. A. DUANE, New York.

ALTHOUGH instances of central scotoma after exposure to sunlight are by no means rare, the subjoined case seems worthy of record, because of the persistence of the scotoma twelve years afterwards, and because of the presence of a peculiar hemiopic and rotating of scotoma scintillans, which apparently was likewise the result of the action of the sun's rays.

The patient, P. W., a man twenty-four years of age, consulted Dr. Knapp on Feb. 5, 1895, and gave the following history: Twelve years previous he had, on the occasion of the transit of Venus,[1] looked directly at the sun through the tube formed by the nearly closed fist. Soon after, he found that when both eyes were open, but not when the left was closed, a greenish cloud hid completely the centre of every object looked at. This had exactly the shape of the illuminated portion of the sun at the time of the transit, *i. e.*, was a circle with a crescentic defect at the upper part corresponding to the spot occupied by the planet at the time. It was then of considerable size, covering an area 5 inches in width when projected upon a surface 15 or 20 inches off. Since then it has gradually decreased in size, but has always retained the same shape. It has always presented the characters of an absolute scotoma, and there has been no interference with color sensations, except that colored as well as white objects were completely blotted out within the area of obscuration. There has been no metamorphopsia.

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[1] In 1882.

Since the date of the transit he has also been subject to attacks in which the entire lower half of the field of vision of the right eye is blotted out, the area of obscuration being covered with dancing light and dark lines. This phenomenon is most distinctly apparent if the left eye is closed, and disappears altogether if the right eye is closed and the left remains open. *In the course of ten or fifteen minutes the obscuration shifts its place so as to occupy successively the temporal, the superior, and lastly the nasal half of the visual field, and then disappears, being succeeded by a dull fronto-temporal left-sided headache lasting for about twelve hours.* The obscuration is absolute, *i. e.*, hides objects completely, and is bounded by a sharp line of demarcation, which is horizontal when the obscured area is situated above or below, and is vertical when this area is situated laterally. These attacks came on at first every four or five weeks, now only every three or four months.

Status præsens.—R E, V $\frac{20}{20}$— (by slight indirect fixation), the letter which is looked at directly being partly cut off by the central spot of obscuration. L E, V $\frac{20}{15}$—. Both eyes emmetropic. Field of vision, R E, presents precisely at point of fixation an absolute, positive scotoma, which at 1 metre distance has a diameter of 2.2 *mm* for white and blue, 2.4 *mm* for red, and 3.0 *mm* for yellow. Outside of these limits no defect in color-sense even by the most delicate tests. Careful examination with the ophthalmoscope, with the pupil dilated by homatropine, showed absolutely no abnormity in the optic disc or macula.

The first point of interest about this case is the length of time for which the scotoma had lasted. In most of the cases collected by MACKAY[1] in his elaborate review of the subject, the injury was comparatively recent (from two days to six months), in two the patient was examined one or two years afterward, and in one (Stigell's) the scotoma was alleged by the patient to exist as long as ten years afterward, although apparently no test was made by the physician to verify this statement.

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[1] *Ophthalmic Review*, Nos. 1, 2, and 3, 1894.

The history of these cases, so far as they have been followed up, is that within a few hours of the time of exposure there is developed a positive, central or paracentral scotoma which varies very greatly in size but which soon begins to diminish both in size and density, until it either disappears altogether or, as in our case and in Stigell's, it is reduced to a very small, but permanent black spot.

The area of retina primarily affected is usually larger—sometimes, as in our case, enormously larger—than the retinal image of the sun. The latter subtends an angle of rather more than ½°, corresponding to a scotoma of 9 *mm* in diameter, at 1 metre. In one case, observed two days after the injury, the scotoma was of just this size, but in other cases during the first week it was from one and a half to two times this size. In the second week the area of retina affected was often reduced to one half or one third the size of the sun's image. After this, the reduction in size seems to take place more slowly. Thus a scotoma, which seventeen days after the injury was 3 *mm* in diameter, was, eighteen months later, about 2 *mm*. The largest scotoma hitherto recorded was in a case of Bock's[1] (= 100 *mm* at 1 metre, corresponding to a diameter of 6°); but the one in the case now under consideration must have originally been much larger still (20° to 25°).

In recent cases at least, the scotoma for colors appears to be greater than that for white, and a color-scotoma may persist after that for white has disappeared.[2] In our case no great disparity between the scotoma for colors and for white any longer exists; and, according to the patient, no such disparity ever existed.

Although neither microscopical observation upon man nor experimental research upon animals has thrown much light upon the pathological changes that underly these symptoms, it is evident from the clinical facts mentioned that these changes must affect that part of the retina upon which the sun's image is cast, and, in addition, a zone of very varying dimensions surrounding and apparently concentric with this area. In the great majority of cases[1] the intensity of the lesion diminishes progressively from the centre to the periphery of the area affected, and, consequently, as the morbid process subsides, the peripheral portions regain their normal character first, so that the scotoma undergoes a concentric contraction. Indeed, so uniform is the latter, that in the few cases that have been observed accurately the scotoma is alleged to have retained pretty exactly its original shape, while all the time growing progressively smaller. In the present case, and in some others in which the scotoma was accurately mapped out,[2] the latter reproduced precisely the form of the non-obscured area of the sun; and it is a remarkable fact that it still retains this shape even when it has shrunk in size so far as to indicate that an area of retina of only one fourth the size of that originally occupied by the sun's image is involved.

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[1] *Centralbl. f. Augenheilk.*, 1890, p. 291.

[2] Mackay, *loc. cit.*, Case IV.

One feature that is often present in cases of this sort, namely, metamorphopsia was absent in our case. On the other hand, the latter was complicated by a very peculiar kind of scotoma scintillans. True scintillating scotoma, with its associated migraine, has not, as far as I am aware, been reported in conjunction with these insolation injuries, and, while the patient was positive as to the fact that this kind of obscuration of sight had not existed previous to his accident, the connection between the two may well be doubted. Nevertheless, the scotoma merits attention not only on account of its possible etiology, but also because of its peculiar character. Occurring as a complete hemianopsia, which, by a process of slow rotation occupied all parts of the visual field in succession, it seems unique even among these curiously varied phenomena. Still more striking is the fact that the hemianopsia is strictly uniocular, disappearing entirely when the left eye is shut. Uniocular hemianopsia is very uncommon, and uniocular scintillating scotoma so extremely rare as to be said by some authors not to exist; yet the patient's statements in this case seem to leave no doubt that the scintillation and hemianopsia actually affected but one eye, and that eye always the same.

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[1] One of Deutschmann's cases (*Archiv f. Augenheilk.* vol. xxviii., part 3, p. 241) is a possible, though not a certain, exception.

[2] Cases IV. and VII. of Mackay's, and probably also one of Deutschmann's, and one of Sulzer's. The data upon this point in the other cases are too vague to be of service. When any are given, the scotoma is usually stated as oval. This oval figure very likely represents a diffusion-image of the more or less crescentic figure afforded by the partially eclipsed sun. At all events this seems to have been so in Mackay's first case, which was myopic, and in which the shape of the scotoma corresponds quite closely to that of the somewhat blurred image which a crescent would form upon the retina of such an eye.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE SECOND QUARTER OF THE YEAR 1894.

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WITH THE ASSISTANCE OF

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Translated by Dr. WARD A. HOLDEN.

Sections I.–V. Reviewed by DR. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, AND TREATISES OF GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL NATURE. STATISTICAL REPORTS.

1. GUAITA and RAMPOLDI. The life and works of Prof. A. Quaglino. *Ann. di Ottal.*, xxiii., 1–2, p. 3.

2. VAN MOLL. Second report of the eye hospital in Rotterdam, 1893.

3. DE HAAS. Twenty-eighth report of the ophthalmic institution in Rotterdam, 1893.

4. BOUVIN. Fourth report of the eye hospital in Gravenhagen, 1893.

5. Gunning. Twenty-first report of the eye hospital in Amsterdam, 1893.

6. Mulder. Report of the eye hospital in Groningen, 1893.

7. Osborne, A. B. The causes of blindness in Ontario. Arch. of Ophth., xxiii., 1, p. 14.

Guaita and Rampoldi (1) present a short biography of Prof. Quaglino, deceased in January last. He was born in 1817, was made assistant to Flarer in Pavia in 1843, and became his successor in the chair of ophthalmology in 1860, which post he held until recently. Between the years 1846 and 1884 Quaglino wrote a great number of papers and brochures on ophthalmological subjects, and made many important translations. The majority of the older Italian oculists of to-day were his pupils. Dantone.

Van Moll (2) in 1893 treated in his hospital 2059 patients, 222 being admitted to the house, and 189 operations were made, among them 54 extractions.

De Haas (3) in 1893 treated in his hospital 4643 cases, 258 being admitted to the house, and 305 operations being done, among them 101 extractions.

In Gravenhagen (4) 4071 patients were treated, 258 admitted, 305 operated, and 101 extractions done.

Gunning (5) in Amsterdam saw 10,990 patients, 678 being admitted; 248 operations were done, among them 27 extractions.

Mulder (6) in Groningen saw 1172 operations, 133 being admitted, 120 operated, and 26 extractions done.

Osborne (7) officially examined the provincial institution for the blind in Brantford with respect to the causes of blindness, and gave a tabular report. Among 132 blind inmates, in 29 the blindness was due to cataract, in 21 to blennorrhœa neonatorum, in 9 to trachoma. Greeff.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

8. Beljarminow. The diffusion in the interior of the eye in different pathological conditions. *Russkaja Medina*, 1894, No. 15.

9. Briggs, W. E. Subconjunctival injections in the treatment of eye diseases. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

10. Report of Committee upon the value of the various objective tests for determining ametropia. *Ibid.*, Sept. 11, 1894.

11. Randall. Retinoscopy as an accurate test for determining errors of refraction. *Ibid.*, Sept. 1, 1894.

12. JACKSON. The visual zone of the dioptric media and its study by means of skiascopy. *Ibid.*, Sept. 1, 1894.

13. STARKEY. Are low-degree lenses "merely of mythical value." *Ibid.*, Sept. 8, 1894.

BELJARMINOW (8) obtained by colorimetric methods on rabbits the following results: 1. Acute processes in the cornea, changing its anatomical structure, increase the diffusion through it. 2. In the cicatrization of wounds the diffusion is increased while the scar is fresh, but is normal in older scars. 3. In cicatricial transformation of the cornea diffusion is greatly diminished. 4. Coloboma of the iris does not affect diffusion. 5. The higher the intraocular tension, the less the diffusion. 6. In aphakia, fluids such as fluorescin diffuse more quickly into the vitreous.

HIRSCHMANN.

BRIGGS (9) has tried the subconjunctival injection of bichloride 1 to 2000, and has found it of most use in syphilitic diseases of the choroid and in ulcers of the cornea. He does not use it in cases of acute iritis. BURNETT.

The Committee (10) (Drs. Jackson, Burnett, Würdemann, and Thompson) appointed to report on the value of the objective tests for the determination of ametropia are of the opinion that no one of the objective tests can be relied upon absolutely. Skiascopy they regard as the most useful single one, since it is more easily applied by the average examiner; that the direct ophthalmoscopic examination gives the refraction of the eye as a whole, while ophthalmometry gives only the corneal astigmatism. Yet all are useful and should be used in every case as corroborators or checks. They regard, however, the objective test, not the letters and test lenses, when applicable, as the final court of appeal. BURNETT.

RANDALL (11) asserts that, while with intelligent patients the subjective tests are the most satisfactory, in doubtful cases skiascopy gives important aid and the most accurate results. By using a plane mirror at 3 or 4 metres he can in favorable cases determine the refraction to within 0.1 D (in extreme cases can recognize a change of 0.05 D), and the axis of astigmatism to within 5°.

JACKSON (12) points out that for accurate results with skiascopy the measurements must be limited to the visual zone, and to this end uses in his examinations a very small point of light, about 5 *mm* in diameter, and the measurement made at ½-metre distance.

BURNETT.

Starkey (13) has accumulated 75 answers to questions as to the value and use of low degree cylinder and spherical lens, sent to various ophthalmic surgeons in the United States, and with only a few exceptions those responding had used them more or less constantly, and with supposed benefit. Burnett.

III.—INSTRUMENTS AND REMEDIES.

14. Baduel. Experimental researches on the action of tincture of iodine injected into the vitreous of a healthy rabbit's eye—a contribution to the study of Schöler's method of treatment in detachment of the retina. *Arch. di Ottalm.*, i., 2, p. 364.

15. De Bono. Suggestive therapy in hysterical affections of the eye. *Ibid.*, i., 12, p. 417.

16. Ciserani. Subcutaneous and subconjunctival injections of cocaine in eye operations. *Ann. di Ottalm.*, xxiii., 1–2, p. 105, and *Arch. di Ottal.*, i., 11, p. 381.

17. Parinaud. Stereoscopy. Model of clinical stereoscopy. *Ann. d'Ocul.*, cxi., p. 406.

18. Baxter, W. E. A new phorometer slide. Arch. of Ophth., xxiii., 1, p. 38.

19. Bumstead. A new centring instrument. *Ibid.*, p. 88.

20. Jackson, E. A triple rotary variable prism. *Ibid.*, p.115.

21. Holden, W. A. On tests of the light sense of the periphery of the retina for diagnostic purposes. *Ibid.*, p. 40.

22. Briggs, W. E. Optico-ciliary neurotomy with demonstration of scissors. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

Baduel (14) has again taken up the subject of injections of iodine into the vitreous of rabbits, and he finds that even very small quantities may be destructive to the tissues, and that injections cannot be recommended as a remedy for detachment of the retina. Dantone.

De Bono (15) reports numerous cases of nervous affections, such as ptosis, amblyopia, etc., in hysterical patients cured by suggestion, both in and out of the hypnotic state.

In the Rosmini Institute, in Milan, all operations, according to Ciserani (16), are done with cocaine. For lid operations subcutaneous injections are made; for iridectomy and cataract extraction instillations into the conjunctival sac; and for tenotomy and enucleation subconjunctival injections; 4 per cent. solution is used. For exenteration of the ball chloroform is given.

Dantone.

PARINAUD'S (17) stereoscope has a larger field than most instruments. The field of each eye extends 50° temporally, giving thus a binocular field of 100° lateral extent. The optical part consists of two convex lenses 4 D, and two prisms 10°. The apex of the prisms may be placed in either direction. The frame of the instrument is adjustable, so that the distance of the images may be varied. The motility of the prisms permits the instrument to be used for practice by squinting individuals.

In the second part PARINAUD calls attention to the fact that stereoscopic vision has been very accurately studied in its geometrical aspects, but less in its physiological. Stereoscopic vision ensues from the fusion of two unsymmetrical incongruent figures, while in binocular vision of objects in a single plane the congruence of the images is a necessary condition of fusion. The incongruence of the fused images in binocular stereoscopic vision is a remarkable feature, which indicates that stereoscopic vision depends upon a psychical process that cannot be geometrically analyzed.

The first condition of stereoscopic vision is that the images to be fused shall fall on identical points in the retinæ. This condition may be obtained without the help of instruments ; it is, however, necessary to relax convergence, which at the same time brings about relaxation of accommodation and blurred images.

One begins by trying to obtain fusion under the easiest conditions, which are later made more difficult by shortening the distance and removing the prisms. SULZER.

BAXTER (18) had two Risley double prisms arranged on the phorometer slide, one vertical and one horizontal, by means of which not only heterophoria can be measured, but also the strength of the recti muscles.

BUMSTEAD (19) describes a new centring instrument having two cells with cross-hairs, which are adjusted before the eyes of the patient so that the cross-hairs before each eye cover a cross in the distance, when the inter-pupillary distance may be read off a scale.

JACKSON'S (20) triple rotary prism consists of two equal prisms which rotate in opposite directions, and combined with these a stationary prism equal to the sum of the other two. With two prisms alone the variable effect increases with the sine of the angle of rotation, and therefore a given amount of rotation produces much less effect near the point where the prisms neutralize each other, than near the point where they produce the maximum resultant effect. The addition of a third neutralizing prism reverses this, and gives a coarser scale near the zero point, which is an advantage.

Groenouw has recently proposed to measure the indirect acuteness of vision by using as perimeter test-objects black points of various sizes on a white ground. The result obtained is termed by him the visual acuteness for points. HOLDEN (21) thinks that the determination of the visual acuteness for points is simply a measurement of the light sense of the periphery of the retina. He has found that tests for the light sense of the periphery in this way often furnish information not obtained by the usual methods. The same results are obtained when black points of various sizes and large gray patches of various intensities are used, and one may be used as a check on the other. The results obtained agree with those obtained by color perimetry, for there cannot be much disturbance of the light sense without affection of color perception, and these tests may often take the place of color tests.

GREEFF.

IV.—ANATOMY.

23. LE DOUBLE. Varieties of the ocular muscles, lids, and eyebrows in the human species. *Arch. d'opht.*, April, 1894, p. 218.

24. BORYSIEKIEWICZ. Further investigations on the finer structure of the retina. F. Deutike, Vienna, Leipsic.

25. DIMMER. On the anatomy and physiology of the macula lutea in man. F. Deutike, Vienna, Leipsic, 1894.

26. BERNHEIMER. The region of the origin of the oculomotor nerve in man. With four colored plates. Bergmann, Wiesbaden, 1894.

27. PFLUEGER. On the lymph circulation in the eye. *Arch. f. Augenh.*, xxviii., p. 351.

After a comparative description of the system of ocular muscles in animals, LE DOUBLE (23) speaks of the abnormalities observed in man, as absence of muscles, superfluous bundles, varieties of origin, alterations in length, thickness, course, and relation to neighboring parts, as well as abnormal attachments ; and he also mentions muscle-bundles which have received particular names, like transversus orbitæ and depressor palp. infer.

He gives two examples of the presence of a retractor muscle in man which had not been previously observed ; a narrow muscular band beginning at the optic-nerve entrance and extending forward between the superior and the external rectus to a point near the junction of the anterior ⅗ with the posterior ⅓ of the circumference of the eyeball. v. MITTELSTÄDT.

BORYSIEKIEWICZ's (24) monograph consists of 64 pages with 65 figures in the text. His views are completely at variance with the hitherto unquestioned observations of Heinrich, Müller, Henle, and other prominent investigators.

DIMMER (25) has made a careful study of the macula of the human retina from fresh eyes, and the interesting results are contained in a monograph of 132 pages. The first part treats of anatomical relations; the second of physiological problems. In a supplement, the views of Borysiekiewicz are sharply criticised and his errors exposed. There are good plates of the sections of the retina through the macula.

BERNHEIMER (26) studied the region of origin of the oculomotor nerve as he previously studied the roots of the optic nerve. The corpora quadrigemina and neighborhood, from 15 brains of the fœtus at various ages, were cut in serial sections in various planes and stained by Weigert's and Golgi's methods. The important findings are shown in a number of colored plates.

V.—PHYSIOLOGY.

28. HELMHOLTZ. On the origin of the right conception of sense impressions. *Zeitschr. f. Psych. u. Phys. d. Sinnesorg.*, vii., p. 81.

29. KOENIG. A previously unobserved form of congenital color-blindness (pseudo-monochromatopsia). *Ibid.*, vii., p. 161.

30. EATON, F. B. The physiology of certain oculomotor symptoms with some recent theories of asthenopia. *Journ. Amer. Med. Assoc.*, Sept. 1, 1894.

31. MADDOX. Latent torsion of the eyes. *Ophth. Review*, xiii., p. 181.

EATON (30) opposes the theory of cyclophoria as advanced by Savage, by showing that the symptoms are physiological and in no way affect complete binocular vision, but, on the contrary, binocular vision would be seriously interfered with by the correction proposed. He also opposes the innervation method of treating insufficiencies of the recti muscles by gymnastic exercises, from the standpoint of physiology and practical experience.

BURNETT.

MADDOX (31) refers to the experiment of Le Conte who was able to prove on himself that outward torsion of the eyes took place during convergence, when the desire for single vision was suspended. This he was able to do by his power of creating at will a strong convergent strabismus. Maddox has devised a method which will enable any one to demonstrate this latent torsion. A prism of 6°, say, is placed apex outwards before the right eye and a vertical line at the near point is observed, the prism is then suddenly rotated to the vertical, and before there is time to fuse the images they can be seen to cross at a small angle. In testing for torsional anomalies at a distance a glass rod 6 inches long and $\frac{3}{4}$ inch thick is held horizontally before the two eyes, a prism is then rapidly rotated as before and enables one to see if the two streaks of light are parallel or slightly inclined to one another. As to the amount of torsion which is necessary to constitute an anomaly the author has no experience. WERNER.

Sections VI–XI. Reviewed by DR. HORSTMANN.

VI—ANOMALIES OF REFRACTION AND ACCOMMODATION.

32. BJERRUM. The determination of acuteness of vision. For students and physicians. Copenhagen, 1894.

33. FUKALA. A contribution to the history of the operative treatment of myopia. *Arch. f. Augenheilk.* xxix., p. 42.

34. VACHER, and FUKALA. On the extraction of the transparent crystalline lens as a prophylactic measure against progressive myopia of high degree and detachment of the retina. *Annal. d'ocul.*, cxi., p. 437.

35. VAN FLEET. Astigmatism and the ophthalmometer. ARCH. OF OPHTH., xxiii., p. 50.

36. PINKHOF. Astigmatism in Amsterdam. *Weekblad*, 1894, No. 23, p. 906.

37. BATES. A suggestion of an operation to correct astigmatism. ARCH. OF OPHTH., xxiii., p. 9.

38. HALTENHOFF. Traumatic paralysis of accommodation. *Soc. franç. d'opht*, 1894.

39. PISCHL, KASPAR. Report on examinations of the eyes of 1900 school-children of the public schools of San Francisco. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

40. SOUTHARD. School-children's eyes—a plea for the ex-

amination of every child's eye when commencing to attend school. *Ibid.*, Sept. 8, 1894.

41. Tiffany, F. B. Anomalies of refraction and of the muscles of the eye. New York, 1894.

Vacher and Fukala (34) reported to the French Ophthalmological Society recent results from the extraction or discission of the transparent lens in myopic eyes. In spite of the good results reported the operation was severely criticised in the discussion by operators who had had less successful results. Sulzer.

Van Fleet (35) examined 100 consecutive patients with the ophthalmometer; 5 had no astigmatism; of the other 190 eyes 177 had regular astigmatism varying from 0.25 — 3. D; 5 of these accepted the full correction, 158 under a dioptry less, and 19 a much weaker cylinder. Such favorable results are not obtained by all writers. The author believes this to be due to lack of skill in using the instrument and to poor instruments.

According to Javal, Hebrews have an astigmatism against the rule which is due to the thick horizontal lines of the Hebrew characters. Pinkhof (36) made a series of observations on this point in Amsterdam and found that there no support could be found for Javal's view and that there was no relation between the astigmatism of a race and its written language.

Haltenhoff (38) describes a case of total bilateral paralysis of accommodation in consequence of a prick with a needle in the breast. Pupils normal. Ruling out traumatic hysteria, we must assume an infectious nuclear change, as after meat poisoning, syphilis, and diphtheria. Other signs of the infection were fever, lasting several weeks, and a recurring eruption on the injured side of the thorax. The traumatic pneumothorax was cured but the paralysis of accommodation persisted.

In his examination, of 1900 pupils of the public schools of San Francisco, Pischl (39) found an increase of myopia with an increase in the grade of the class going from 3.98% to 11.59%, the latter being in the normal school. As to nativity, 25.25% are of American parentage, 49.50% of European, and 25.25% mixed.

Burnett.

This paper of Southard's (40) is rather a plea for the hyperope who should have assistance in the beginning of his school life. He found in his examination of the students of the University of California 68% of refractive errors, and of these only 6% were myopic; and in 1300 cases of refractive error, 75% were hyperopic.

Burnett.

VII.—LIDS.

42. ZIMMERMANN, M. W. Primary melanotic sarcoma of the eyelid, with report of a case. *Ophth. Rev.*, xiii., p. 184.

43. GERMAIN. The operation for distichiasis. *Soc. franç. d'opht*, 1894.

44. PESCHEL. A modification of the ptosis operation. *Centralbl. f. prakt. Augenheilk.*, 1894, p. 306 (a modification of Birnbacher's operation).

ZIMMERMANN'S (42) patient was a healthy man aged fifty. One sister had an abdominal tumor, otherwise family history was good. Ten months before, he noticed a small growth at the outer side of the left upper lid. It grew very slowly until one month before admission when it increased rapidly. There was no recurrence eighteen months after removal. The tumor was a melanotic spindle-celled sarcoma. The author could only find two cases on record. Rapid growth, peculiar dark color, with no hemorrhage to account for it are the main points in the diagnosis.

WERNER.

VIII.—LACHYRMAL APPARATUS.

45. ADLER. A case of bilateral mumps of the lachrymal gland. *Wiener med. Wochenschr.* 1894, No. 14.

46. KALT. Follicular dacryocystitis. *Soc. franç. d'opht.*, 1894.

ADLER'S (45) case was a man of eighteen, in whom there was marked nodular swelling of both lachrymal glands, with simultaneous swelling of the sublingual, submaxillary, and cervical glands.

At an autopsy, KALT (46) discovered by accident a follicular inflammation of the mucosa of the lachrymal sac, with normal bulbar and palpebral conjunctiva. The mucosa was 1 *mm* thick in consequence of a diffuse infiltration with round cells. The cylindrical epithelium, which contained numerous goblet cells, is wanting in places, leaving the infiltration exposed. SULZER.

IX.—MUSCLES AND NERVES.

47. FERRI. Concomitant strabismus in its various forms, with special reference to its etiology. *Annal. di Ottalm.*, xxiii., p. 59.

48. DUANE. The treatment of heterophoria. An answer to Dr. Gould. *Annals of Oph. and Otol.*, 1894, July.

49. CHEVALLEREAU. Astigmatism and convergent strabismus. *Soc. franç. d'opht.*, 1894.

50. MADDOX. Latent torsion of the eyes. *Ophth. Rev.*, xiii. p. 181.

51. PRICE. G. H. Cyclophoria, its diagnosis and treatment. *Journ. Amer. Med. Assoc.*, Sept., 8, 1894.

52. THOMAS. Muscular asthenopia and its treatment by graduated tentomies. *Trans. Med. Soc. of Pennsylvania*, 1894.

53. BALL. Advancement of the recti muscles by the folding method. *Ther. Gaz.*, April, 1894.

54. EULENBURG. A case of isolated traumatic basal paralysis of the abducens nerve. *Neurol. Centralbl.*, No. 16, 1894.

55. DUANE. Paralysis of the superior rectus and its bearing on the theory of muscular insufficiency. ARCH. OF OPHTH., xxiii., p. 61.

56. GAZÉPY. Two cases of congenital external ophthalmoplegia. *Arch. d'opht.*, xiv., 5, p. 77.

57. GRADLE. Paralysis of the ocular sympathetic nerve fibres. *Ann. of Ophth. and Otol.*, July, 1894.

58. PARENTEAU. Two observations of intermittent muscular paralysis. *Soc. franç. d'opht.*, 1894.

In this paper DUANE (48) advances arguments against the acceptance of the theory that a faulty innervation is the cause of all cases of heterophoria as has been recently advanced anew. He does not believe there is a single cure-all for heterophoria, but that some are relieved by correction of the ametropia, some by exercise of the muscles and some by tenotomies and some apparently not by any means we have yet at command. BURNETT.

CHEVALLEREAU (49) in 200 cases of squint found over a half-dioptry of astigmatism in 119 cases. In 46 cases both eyes were equally astigmatic, while in the great majority of cases the deviating eye had the higher degree of astigmatism. In 8 cases of alternating squint, the eye usually fixing had the higher degree of astigmatism. The author believes that astigmatism as well as hyperopia may predispose to squint. SULZER.

Among other facts brought forward in PRICE'S (51) paper, which deals with the plan of Savage for the selection and treatment of insufficiencies of the oblique muscles by cylindrical glasses, is that in 200 tabulated cases there was insufficiency of the superior oblique in 25 per cent. and of the inferior oblique in ½ per cent.

BURNETT.

From a considerable experience in making tenotomies, extending over six years, THOMAS (52) is convinced that it is possible to make a gain of from 1° to 8° without a complete severance of the tendon. BURNETT.

In the operation of advancement, BALL (53) first makes a tenotomy of the antagonist; he then makes an incision through the conjunctiva and subconjunctival tissue over the insertion of the muscle to be advanced, and passes a squint hook beneath it. After detaching the muscle from the subjacent tissue, two needles are passed through the muscle that is folded posterior to the hook, and are passed out through the conjunctiva, one above, the other below the cornea. After removing the hook, the threads are knotted and the conjunctival wound closed by a superficial suture.

EULENBURG (54) reports a case of complete paralysis of the right abducens after a stab wound in the right temple, in a man of thirty-two. There were headache, giddiness, vomiting, and psychical disturbances. The abducens was no doubt wounded at the place where it is isolated from the other ocular nerves, viz., in the region where it runs toward the clivus basilaris behind the dorsum sellæ to enter the cavernous sinus, and in the latter at the outer side of the internal carotid.

DUANE (55) is of the opinion that isolated paralysis of the superior rectus is not so rare as was formerly believed. He finds it frequently. It is always bilateral, and is found in young individuals who use the eyes continually in near work. He believes that the paralysis develops from a congenital weakness or anomaly of the muscle. GREEFF.

GAZÉPY'S (56) patients were a brother and sister, aged twenty-five and eighteen, of healthy parentage and with healthy brothers and sisters. In the man the superior and external rectus of both eyes were paralyzed and the inferior rectus of the left; in the girl the superior rectus of both eyes, the right internal and the left external rectus. In both cases there were ptosis and lagophthalmus from paralysis of the lower lid, but the pupillary reaction and accommodation were normal. Both patients were weak intellectually, and had other nervous defects.

v. MITTELSTÄDT.

GRADLE (57) gives the histories of three cases of paralysis of the ocular sympathetic nerve fibres. There were the usual symptoms of narrowing of the palpebral aperture, pupillary contraction, increased vascularity, but no pain. There was no cause discovered in any of the cases. Most of the cases had existed for a long time, and no remedial measures were found beneficial.

BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

59. HIRSCH, C. On orbital phlegmon. *Prager med. Wochenschr.*, 1894, Nos. 14–20.

60. MAY. A case of orbital cellulitis after a tenotomy for squint. *Annals of Ophth. and Otol.*, July, 1894.

61. FRIEDENWALD and CRAWFORD. Exophthalmus due to orbital hemorrhage. ARCH. OF OPHTH., xxiii., p. 142.

62. MARTIN. Tumors of the orbit caused by frontal-sinus affections. *Soc. franç. d'opht.*, 1894.

63. DE BONO. A case of orbital osteoma. *Arch. di Ottalm.*, i., p. 304.

64. DE VINCENTIIS. On exophthalmus from orbital neoplasms. *Clin. Ocul. di Napoli*, iv., 1.

65. BAYER. On pulsating exophthalmus. *Prager med. Wochenschr.*, No. 28, 1894.

66. OGILVY. Notes of a case of traumatic enophthalmus. *Ophth. Rev.*, xiii., p. 145.

67. RAMAGE. A case of congenital anophthalmus. *Brit. Med. Jour.*, March, 1894.

68. GROHMAN. Contributions to the etiology and symptomatology of Basedow's disease. *Inaug. Dissert.*, Berlin, 1894.

69. EULENBURG. Basedow's disease and the thyroid gland *Deutsch. med. Woch.*, 94, No. 40.

MAY'S (60) patient was a boy of six years who had a strab. conv., for which the left internus was divided, and later the right. After the latter operation a typical orbital cellulitis set in, and pus discharged through the conjunctival wound. In a little over a month the eye was quiet, but there was some optic-nerve atrophy and a divergent squint. The operation was done under aseptic conditions.

BURNETT.

Exophthalmus from hemorrhage may be spontaneous or traumatic. Both are rare. FRIEDENWALD and CRAWFORD (61) describe the cases previously reported and add a new one. In their case there was spontaneous orbital hemorrhage in a negro addicted to drink.

GREEFF.

MARTIN (62) reports histories of cases which show that empyæma of the frontal sinus may bring about a swelling of the periosteum of the orbit that compresses the optic nerve and leads to visual disturbance of various degree. The compression may be localized and only affect the inner upper segment of the nerve. Catheterization of the frontal sinus is easy when there is empyæma, and is often necessary in order to confirm the diagnosis.

SULZER.

DE BONO (63) reports a case in which an osteoma, situated in the nasal wall of the orbit, was successfully removed with preservation of sight.

DANTONE.

DE VINCENTIIS (64) gives a careful and detailed study of exophthalmus from neoplasms, hemorrhage, and aneurysms.

DANTONE.

OGILVY (66) records the particulars of a case of traumatic enophthalmus from Fuchs's Clinic. The patient had been knocked down and trampled on by an ox. The left eye was dislocated inwards and downwards, so that the finger could be passed between it and the orbital roof. The left orbit measured 0.5 *cm* more, in the vertical, and 0.5 *cm* less, in the horizontal, than the right. The malar bone was crushed in towards the nose, but there was also a fracture of the lower margin of the orbit extending into the infra-orbital canal. This was believed to be the cause of the enophthalmus. The eye was ruptured and hopelessly injured. Abstracts of recorded cases are given (also in Beers's paper on the same subject, in these ARCHIVES).

WERNER.

RAMAGE'S (67) patient was seen when aged six months. The left eye was normal, the right being replaced by a small fibrous stump. The ocular appendages were normal.

GROHMAN (68) describes thirteen cases of Basedow's disease, four of which died, while the rest were improved.

According to EULENBURG (69) the thyroid gland, in its normal activity, changes the composition of the blood in a peculiar way. In Basedow's disease there is a progressively increased secretion from the gland, with possibly a qualitative change in the secretion by which the latter is given a specific pathogenic quality. This increased and altered secretion may be due to increased arterial congestion, or to the blood changes preceding or accompanying Basedow's disease.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

70. WESTHOFF. Conjunctivitis neonatorum. *Weekblad*, 1894, p. 174.

71. REICH-HOLLANDER. The external use of quinine in blennorrhoic ophthalmia. ARCH. OF OPHTH., xxiii., p. 30.

72. UHTHOFF. A further contribution to diphtheritic conjunctivitis. *Berl. klin. Wochenschr.*, 1894, Nos. 34 and 35.

73. ALBRAND. On pemphigus of the conjunctiva. *Zehender's klin. Monatsbl.*, xxxii., p. 220.

74. SACHSALBER. Pemphigus of the conjunctiva. *Ibid.*, p. 241.

75. HERTER. Syphilitic papules of the bulbar conjunctiva. *Ibid.*, p. 200.

76. GIESE. Measurements of the temperature in the human conjunctival sac. *Arch. f. Augenheilk.*, xxviii., p. 292.

77. BERRY. On a rare form of bullous conjunctivitis. *Ophthalm. Rev.*, vol. xiii., p. 81.

78. GIFFORD. The shady side of the surgical treatment of trachoma. *Med. Rec.*, Aug. 25, 1894.

According to WESTHOFF (70), the treatment of conjunctivitis of the new-born consists in the frequent cleansing with cotton dipped in solutions of boric acid, chlorine, or permanganate of potash, and the instillation once or twice daily of 1 % silver solution. WESTHOFF.

REICH-HOLLANDER (71) has used a solution of quinine in blennorrhœa with good results. His solution is quin. sulph. 2, acid mur. dil. 0.75, aq. 180. This is used for washing out the conjunctiva every hour, iced application being used simultaneously.

GREEFF.

UHTHOFF (72) describes four cases of diphtheritic conjunctivitis which ran their course under the clinical picture of a relatively slight benign conjunctivitis. In three cases true virulent diphtheria bacilli were demonstrated, and in each case other members of the family or neighbors had faucial diphtheria.

ALBRAND (73) reports a case of pemphigus of the conjunctiva in a girl of seventeen, who had suffered from the same trouble in her eighth year. On both sides the conjunctiva shrunk until there was complete symblepharon, and there was opacity or perforation of the cornea.

SACHSALBER (74) observed the same disease in a woman of sixty-three, which led to shrinking of the conjunctiva and opacity of the cornea.

HERTER (75) describes a case in which two syphilitic mucous papules appeared on the bulbar conjunctiva above and below the cornea in one eye. These disappeared under the inunction treatment.

GIESE (76) made a series of temperature measurements in the conjunctival sac of healthy and inflamed eyes, and observed the effect of cold and warm applications. He found, in opposition to Silex, that cold applications reduced the temperature, while warm ones increased it.

BERRY'S (77) patient, a woman aged forty-seven, complained of discomfort after reading. The conjunctiva of each upper lid presented an irregular stellate cicatrix. The history was that a year previously the eyelids had been swollen for three weeks and that she could feel like little "bladders" under them with her finger, at the same time she had an attack of dermatitis herpetiformis. The scars in the conjunctiva were quite different from those of pemphigus. WERNER.

GIFFORD (78) utters a note of warning in regard to the mechanical treatment of trachoma, citing several cases in which corneal ulcerations have developed after such operations, which ended in blindness. [Corneal ulceration from mechanical treatment has never come to my knowledge, though I have treated hundreds of cases.—H. KNAPP.] BURNETT.

79. DOLSCHENKOW. Lactic acid in corneal ulcers. *Wjestnik. Ophth.*, xi., 3, p. 73.

80. GALLENGA. On a rare form of progressive opacity of the cornea (sclerosis with hyalin degeneration). *Arch. di Ottalm.* I. 12, p. 385.

81. KUTHE. Clinical contributions to the study of neuroparalytic keratitis. *Centralbl. f. prakt. Augenh.*, 1894, p. 300.

82. BAAS. On congenital corneal affections. *Ibid.*, p. 289.

83. BOURGEOIS. Conjunctival autoplasty in a case of corneal fistula. *Rev. d' opht.*, 1894, 3, p. 156.

84. BERGER. Keratomalacia consecutive to infectious diseases. *Bull. méd.*, 1894, April 4.

In obstinate corneal ulcers with pain and photophobia, in threatening crescentic ulcers, and in infected ulcers DOLSCHENKOW (79) recommends touching the ulcer with lactic acid. The rapid and favorable results are similar to those following the use of the thermo-cautery. HIRSCHMANN.

GALLENGA (80) gives a detailed clinical and histological description of sclerosing of the corneal tissue with hyalin degeneration in both eyes of a young girl. DANTONE.

In a fireman of forty-one after a severe contusion of the head, KUTHE (81) saw develop a neuroparalytic keratitis in the right eye, with paralysis of the 3d, 6th, olfactory, and hypoglossal nerves.

In two members of a family BAAS (82) found congenital opacity of the cornea, in one case the cornea appearing as a sclerotic broadening of the limbus, in the other the appearance resembling parenchymatous keratitis. The iris showed some atrophy and anterior synechiæ.

In BOURGEOIS'S (83) case a lime burn was followed by a corneal fistula above and to the nasal side. A flap of conjunctiva 5 *mm* broad and 15 *mm* long was loosened above the fistula, its upper end brought down and turned so that its raw surface lay in contact with the fistula, and then fixed by two sutures through the divided conjunctiva near the limbus.

Sections XII.–XXI. Reviewed by DR. SILEX.

XII.—IRIS.

85. STÖRMANN. On eye inflammations from the penetration of caterpillar hairs. *Inaug. Dissert.*, Berlin, 1894.

86. VELHAGEN. A case of iris tuberculosis. *Zehender's Klin. Monatsbl.*, xxxii., p. 121.

87. ROSENZWEIG. A case of congenital serous cyst of the iris. *Beiträge zur Augenheilk.*, xiv., p. 34.

88. SNELLEN, H., Jr. On the etiology of Descemetitis. *Weekblad*, 1894, p. 849.

To the observations of Pagenstecher, Weiss, Krüger, and others, STÖRMANN (85) adds the report of a number of cases of inflammation caused by caterpillar hairs, seen in the Berlin University eye clinic, and mentions seven species of caterpillar that had caused inflammation.

VELHAGEN (86) found that the iris was the first tissue to be affected by tuberculosis in his case of a boy of eight. Anatomical examination showed that Fontana's spaces favor the extension of tuberculosis, for in its inner half the iris was for the most part preserved, while in the outer angle tubercles had developed. The first colonization of bacilli had perhaps taken place in Fontana's spaces.

In ROSENZWEIG's (87) case of cyst in the iris in an infant of three months, both the anterior and posterior wall were formed of iris which was simply split into two sheets. The cyst wall was lined with stratified epithelium. Epithelial cysts, not traumatic in origin, may develop in various parts of the body, like the dermoids from displacement of the epiblast.

Although it is generally believed that Descemetitis is due to the deposit of the inflammatory products of iritis serosa and cyclitis, SNELLEN (88) found in two cases that the precipitate consisted chiefly of living bacteria. In the first case bacteria alone were present ; in the second, possibly a later stage, the bacteria were mixed with fibrin and leucocytes. He believes the development of these bacteria and the deposit from the aqueous humor on the membrane of Descemet to be the primary affections, and that the toxic elements so produced cause the serous inflammation of the iris. WESTHOFF.

XIII.—CHOROID.

89. MITVALSKY. On choroidal tumors. *Arch. f. Augenheilk.*, xxiii., 4, p. 323. (Will be translated.)

90. TAILOR. Cavernous angioma of the choroid. *Ann. di Ottalm.*, xxiii., 1–2, p. 51.

91. VEILLON and MORAX. Suppurative choroiditis due to streptococci, supervening spontaneously on a medical septicæmia with suppurative arthritis. *Ann. d'Ocul.*, cxi., p. 341.

92. BATTEN. Localized posterior staphylomata with distortion of the retinal vessels. *Ophth. Rev.*, xiii., p. 113.

In an eye enucleated for unbearable pain, a diagnosis of cyclitis or choroidal neoplasm being made, TAILOR (90) found a cavernous angioma of the choroid which had involved the posterior and inferior portion of that membrane. DANTONE.

The general symptoms of the case of septicæmia observed by VEILLON and MORAX (91) were those of an acute articular rheumatism, and the ocular affection was a spontaneous purulent iridochoroiditis of the left eye. The enucleation of the eye and the opening of the left knee-joint furnished material for bacteriological examination, and streptococci were obtained in pure culture

SULZER

Batten (92) points out that, in myopia the bulging of the sclerotic may occur at other parts of the fundus besides the macula lutea, and the retinal vessels then become distorted in the direction of the staphyloma. The refraction of the different portions of the fundus should be tested. Staphylomata may occur: (1) in the temporal region beyond the macula; (2) in the fundus to the nasal side of the disc; (3) below the disc; and lastly and most rarely above the disc. The vessels are drawn toward the staphylomatous region. Staphylomata near the macular region diminish the visual acuity more than a staphyloma of the macula itself. Werner.

XIV.—GLAUCOMA.

93. Warlemont. Congenital buphthalmus with preservation of good vision in a patient of thirteen. *Soc. franç. d'opht.*, 1894.

94. Snellen. The treatment of infantile glaucoma leading to buphthalmus. *Weekblad*, 1894, p. 175.

95. Fromaget. Intraocular hemorrhage from a sarcoma of the choroid after instillations of atropine. Glaucomatous accidents. *Rev. génér. d'ophth.*, 1894, No. 4.

96. Hosch. Glaucoma with extensive hemorrhages and peculiar changes in the retina. *Arch. f. Augenheilk.*, xxviii., p. 316.

97. Renneke. Glaucoma in aphakic eyes. *Inaug. Dissert.*, Berlin, 1894.

In Warlemont's (93) patient the eyeball was enlarged in all dimensions, the cornea, which was clear, measuring 17 *mm* in diameter; the iris was tremulous, the pupil excentric, large, but responsive, and the vision $\frac{2}{3}$. Sulzer.

According to Snellen (94) there are two forms of glaucoma, anterior and posterior. Glaucoma anterius occurs with iritis, keratitis diffusa, Descemetitis, and very typically with buphthalmus. Anatomically the spaces of Fontana are poorly developed in buphthalmus. Iridectomy is dangerous on account of the great collapse. Small and repeated sclerotomies with the use of miotics are of advantage in keeping the tension normal and preventing further increase. Westhoff.

Fromaget's (95) case was that of a woman of fifty-six, with a white fascicular sarcoma in the left eye. The day following the instillation of atropin glaucomatous symptoms developed. When the patient returned a week later there was a dark intercalary staphyloma beneath the upper lid. The large sarcoma was found to be imbedded in a mass of blood. Fromaget believed the hemorrhage and increase in tension to be due to the use of atropine. SULZER.

XVII.—LENS.

98. SCHIÖTZ. Preparatory treatment of cataract patients. *Norsk. Mag. f. Lageredsk.*, No. 4, p. 36, Christiania, 1894.

99. SIMI. A discourse on the operation for cataract. *Boll. d'Ocul.*, xvi., 1–3, 5.

100. MÜLLER. Has lenticonus its cause in an anomaly of the posterior surface of the lens? *Zehender's klin. Monatsbl.*, xxxii., p. 178. (Two cases.)

101. FAGE. Simple extraction in atropinized eyes. *Soc. franç. d'opht.*, 1894.

102. HALTENHOFF. Treatment of traumatic cataract. *Soc. franç. d'opht.*, 1894.

103. DOLGANOFF. On the changes in the corneal astigmatism after the extraction of cataract. *Arch. f. Augenheilk.*, xxix., p. 13.

104. SMITH. Morphine hypodermically as a means to prevent prolapse of the iris in simple extraction. ARCH. OF OPHTH., xxiii., p. 85.

SCHIÖTZ (98) recommends the daily washing of the conjunctival sac with one-half per cent. boiled salt solution four or five days before the operation. The lashes are then pulled out, which is not a painful or irritating process in old people. It is often difficult to say when the eyes are free from irritation; it is therefore advisable when the eye appears normal to bandage it twelve to twenty-four hours, any secretion of mucus being then discoverable. So long as the eye does not remain white and free from mucus under this trial bandage Schiötz does not operate. SCHIÖTZ.

SIMI (99) discusses the various steps of cataract extraction, the advantages and disadvantages of iridectomy in respect to the removal of the lens, prolapse of the iris, infection of the wound, and the resultant vision, and concludes that methodical excision of the iris is to be avoided. DANTONE.

FAGE (101) recommends atropinization as a preparatory measure in cataract extraction. Prolapse is not rendered more frequent, and the dilated pupil facilitates the operation, allowing a more thorough removal of cortical masses. SULZER.

HALTENHOFF (102) discusses the present status of the treatment of traumatic cataract. De Wecker states that traumatic cataract heals spontaneously when there is no infection. All treatment should therefore be abstained from. "A simple puncture of the lens-capsule suffices, when done aseptically, to bring about the complete absorption of the lens even in old people. As soon as infection takes place, the absorption of the lens ceases, even when the capsular opening is wide and the patient young."

SULZER.

DOLGANOFF (103), after extractions with the Graefe knife upward, found with the Javal ophthalmometer an average astigmatism of 4.9 D two weeks after operation, 2.8 D three to four weeks after, and 2.6 D five to six weeks after operation. The operation causes a decreased refraction in the vertical meridian, and an increased in the horizontal. Later the vertical increases and the horizontal diminishes. He finds that the horizontal increase affects the degree of astigmatism more than the vertical decrease in refraction, as was formerly supposed.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

105. MACKAY. On blinding of the retina by direct sunlight. *Ophth. Rev.*, xiii., p. 1, 41, 83.

106. HILBERT. Subjective color sensations due to the action of toxic substances. *Arch. f. Augenh.*, xxix., p. 29.

107. HEINZEL. Transient blindness in the period of lactation. *Beiträge zur Augenh.*, xiii., p. 13.

108. ROTHMANN. On transient blindness from uræmia. *Berl. klin. Wochenschr.*, 1894, p. 691.

109. DESPAGNET. Infectious retinal arteritis of both eyes; paludism. *Soc. franç. d'opht.*, 1894.

110. WESTHOFF. A peculiar case of varicose dilatation of the retinal veins. *Centralbl. f. prakt. Augenh.*, xviii., p. 166.

111. CHAUVEL. Ophthalmological studies. Hemeralopia. *Rec. d' opht.*, 1891, No. 1.

112. ROMANO-CATANIA. On the nature of hemeralopia. *Arch. di Ottalm.*, 1, 8–9, 257.

113. RANDOLPH. The significance of albuminuric retinitis in pregnancy. *Johns Hopkins Bulletin*, June–July, 1894.

114. GROENOUW. Contributions to the study of concentric contraction of the visual field. *Graefe's Archiv*, xl., 1, p. 173.

115. ARMAIGNAC. A case of spontaneous cure of detachment of the retina. *Soc. franç. d'opht.*, 1894. (Rupture of the sclera of a boy of nine by an arrow; detachment of the retina in the region of the wound; permanent reattachment in three weeks.)

116. CARTER. A case of rare and fatal disease of infancy with symmetrical changes in the yellow spot. ARCH. OF OPHTH., xxiii., p. 126.

117. WOLFE, J. R. On the treatment of detachment of the retina. *Brit. Med. Journ.*, April, 1894, p. 848. (A case improved by scleral puncture.)

118. STRAUB. Solutio retinæ. *Weekblad*, 1894, part ii., p. 181.

MACKAY'S (105) very interesting paper consists of three parts, viz.: (1) an historical survey containing abstracts of recorded cases; (2) a description of seven new cases of "Eclipse Blinding"; (3) a résumé of the experiments of Czerny and Deutschmann; observations on the pathology, symptoms, prognosis, and treatment of the disease occupying the remaining pages, After a momentary glance at the sun with unprotected eyes, an after-image persists for some time, undergoing rapid alterations in color; but if, according to the author, the retinal stimulation surpasses the physiological limit, the after-image does not go through different phases of color. In four cases out of the seven the positive scotoma was not noticed until one or two hours had elapsed; it varied in size from 3 to 20 *mm*, at a distance of 1 *m*. It was described as "a gray spot," "a luminous disc," or "revolving star." The defect for colors extended over a larger area than for white. The peculiar revolving movement, or oscillation, which was sometimes seen in the scotoma, is usually very persistent and was noted in one case 2½ years after the accident. It has never been recorded in any case with V less than ⅓. Metamorphopsia was complained of spontaneously in one case; macropsia persisted in four cases and micropsia in one,—in fact only one patient recovered completely. The ophthalmoscopic appearances were sometimes normal; when changes were noted, in the first week, they consisted in loss of light reflex at the macula, or in more severe cases, a small pale orange spot near the fovea. There may also be some darkening of the fundus at the *m. l.* In the second week, the pigmentation tends to increase. After three weeks or a month the fundus usually appears normal. The silvery-white patch at the fovea, which occurred as the result of experiments on animals, has not been observed in man. As regards prognosis, it may be remarked that no patient with V less than $\frac{1}{3}$ has regained $\frac{6}{6}$. WERNER.

HILBERT (106) gives first a brief summary of the literature of subjective color sensations after the use of toxic agents. Hyoscyamus niger causes erythropsia, santonin a yellow sensation. After the use of picric acid the author observed sensations of yellow lasting two hours, and all shades of green appeared yellowish-green, while the perception of other colors was not much affected, the action of picric acid thus differing from that of santonin. Pencilling the feet with chromic-acid solution caused sensations of yellow, and some fungus-poisons cause sensations of violet.

HEINZEL'S (107) case was a woman of twenty-five who became blind eight weeks after parturition. Ophthalmoscopically there was papillitis. Diaphoretic treatment improved the vision, but the field remained concentrically contracted. The author supposed that there had been a retrobulbar neuritis from the toxic products in the blood.

ROTHMANN'S (108) nephritic patient became blind suddenly in the right eye. The left was normal. Both pupils were of medium width, the right irresponsive to light but reacting consensually, the left responding to light but not reacting consensually. Some days later there was transient blindness in the left eye. Later the vision became normal, and the ophthalmoscopic appearance was normal. Anatomically there was found a slight thickening of the optic nerve sheaths and of the walls of the smaller vessels of the nerve. Rothmann draws the following conclusions: Blindness from uræmia is of peripheric origin caused by œdema of the nerve sheaths. This is also the case in amaurosis after hemorrhages. The presence of the pupillary reaction does not rule out compression of the optic nerve. Preserved pupillary reaction is a favorable symptom as regards vision, but the prognosis is not absolutely unfavorable with irresponsive pupil. Too great pressure may lead to degeneration of the nerve fibres and permanent blindness. In transient blindness the optic nerve is later normal or only degenerated in the periphery.

From 70 detailed observations of hemeralopia, CHAUVEL (111) endeavors to explain the etiology of the affection, but his conclusions are all negative. In some cases general diseases were present, but the author regards their connection with the ocular inflammation as questionable. SULZER.

The lately deceased ROMANO-CATANIA (112) regarded hemeralopia as a temporary or permanent anomaly of adaptation of the retina.

RANDOLPH (113) gives the analysis of five cases of albuminuric retinitis in pregnancy coming under his observation and the report of the pathological conditions found in the retina of one case. These were: 1. Great œdema of the entire retina, and as a consequence increased depth of the retina. 2. Hyperplasia of the neuroglia, especially in the nerve-fibre layer. 3. Presence of hyaline masses throughout the entire retina, most pronounced in the external molecular layer, and the formation of spaces. The changes were most striking near the disc. BURNETT.

STRAUB (118) made two parallel incisions in the sclera, at the site of the retinal detachment, with a stop-needle, and enlarged the wounds with a flat stilet, after dissecting up the tissues from the sclera. The conjunctival wound was then closed, and through it, with a Pravaz syringe, were injected near the scleral wounds two drops of sublimate 1:5000 with cocaine. The injection was repeated three days later. In a week the retina had become reattached and the visual field was nearly normal. WESTHOFF.

XIX—OPTIC NERVE.

119. WESTHOFF. An unusual ophthalmoscopic picture. *Wochbl.*, 1894, ii., p. 173.

120. VIGNES. Retrobulbar neuritis from late hereditary syphilis. *Soc. franç. d'opht.*, 1894.

121. MAUTHNER. Tendency to reparative action in the eye. *Wien. med. Blätter*, 1894, Nos. 17 and 18.

122. DOLGANOFF. An unusual case of very deep physiological excavation. *Wjest. Ophth.*, 1894, No. 3; and *Arch. f. Augenh.*, xxviii., p. 348.

123. KOENIG. Hereditary atrophy of the optic nerves. *Soc. franç. d'opht.*, 1894.

124. MALGAT. Confirmed atrophy of the optic nerves. Papillæ white. Treatment with strichnine. *Rec. d'opth.*, 1894, No. 5.

125. TAILOR. Endothelioma of the sheath of the optic nerve. *Ann. di Ottalm.*, xxiii., 1–2, p. 18.

126. SNELL. Remarks on ambylopia from di-nitro-benzol. *Brit. Med. Journ.*, March, p. 449.

127. MITCHELL, S. Two cases of lesion of the optic tracts from la grippe. *Ophth. Rec.*, Aug., 1894.

WESTHOFF'S (119) case was a girl of eighteen with $V = \frac{6}{8}$ in the emmetropic right eye. Color perception normal. With the perimeter a triangular defect was found with its apex in the blind spot. To the nasal side of the disc was a triangular coloboma sharply limited above but less sharp below. From above, inward, and below very tortuous veins ran to the disc, one of them showing varicose dilatations. WESTHOFF.

VIGNES'S (120) patient was a boy of thirteen who became perfectly blind in the course of a few days with a central scotoma. The disc was blurred and infiltrated, the veins tortuous, the arteries narrow. The child's father was syphilitic. The sight was restored under mercurial treatment. SULZER.

MAUTHNER (121) reports a case of a boy of fifteen, blind from a neuritis caused by chronic inflammation at the base of the skull after injury. Later the boy had an attack of variola and sight gradually returned to the normal, there remaining, however, a defect in the field above.

A child of five lost sight and hearing from meningitis ; atrophy of the optic nerves and retinal vessels. A diagnosis of exudation on the base of the skull was made ; eight weeks later the vision was good with marked concentric contraction of the field. Probably the central artery of the retina was compressed, and only the fovea, nourished by a choroidal vessel, continued to functionate.

DOLGANOFF'S (122) patient, aged thirty-nine, had R. myopia 1. D, $V = \frac{20}{20}$; L. E., $V = \frac{20}{20}$. The very deep excavation occupied the temporal $\frac{2}{3}$ of each disc, and had a depth corresponding to a correcting glass of 8 D. HIRSCHMANN.

MALGAT (124) observed a case in which the discs were white and the retinal vessels narrow, but in which a certain amount of vision was obtained. The vessels also became fuller. SULZER.

An orbital tumor probably of traumatic origin which led to blindness and exophthalmus was removed by De Vincentiis, and the microscopic examination by TAILOR (125) showed it to be an endothelioma arising from the nerve sheath. Although the nerve had been involved almost to its point of entrance, no recurrence took place. DANTONE.

SNELL (126) studied the effects of di-nitro-benzol on workers in an explosive factory. The ocular symptoms observed were : Failure of sight, often to a considerable degree, in a more or less equal extent on the two sides ; concentric contraction of the visual field, with, in many cases, central color scotoma ; enlargement of retinal vessels, especially the veins, even in cases where there was no eye complaint ; some blurring, never extensive, of the edges of the disc, and a varying degree of pallor. Cessation of work, with benzol, leads to recovery. The general symptoms were : Asphyxiated and anæmic appearance, pulmonary murmur, and breathlessness. Urine sometimes black in color ; the blood was often thin and dark-colored. Nervous system : Giddiness, headache, delirium, numbness, loss of sensation, weakness in the limbs. Patellar reflexes normal or exaggerated. WERNER.

MITCHELL (127) reports two cases of optic-nerve lesion following grippe. Case 1 : A woman of twenty-two, soon after an attack of la grippe, was affected with left hemianopia. The only change noted in the fundus was a diminution in the calibre of the retinal vessels. Case 2, also a woman, had a pronounced choked disc, first in the right and later in the left eye, coming on immediately after an attack of grippe. Visual acuteness reduced very low, but the final outcome was $\frac{20}{20}$ in both. BURNETT.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

128. HIRSCHBERG. On the removal of iron splinters from the retina. *Deutsh. med. Wochenschr.*, 1894, Nos. 23 and 25.

129. SULZER. Adaptation of the electro-magnet for the removal of iron splinters from the eyeball. *Report of 11th Internat. Congress ; Arch. f. Augenh.*, xxix., p. 55.

130. DEUTSCHMANN. Extraction of an iron splinter from the vitreous by means of a strong magnet. *Beiträge zur Augenh.*, xiii., p. 98.

131. HAAB. A new electro-magnet for the removal of iron splinters from the eye. *Ibid.*, xiii., p. 68.

132. GALLEMAERTS. The location of foreign bodies in the eye by means of Girard's magnetometer. *Soc. franç. a'opht.*, 1894.

133. HIRSCHBERG. Shot injury. *Berliner klin. Wochenschr.*, May, 1894.

134. HÜRZELER. On the employment of electro-magnets for the extraction of iron splinters from the eye. *Beiträge z. Augenh.*, xiii., p. 20.

135. KNAPP. On the tolerance of foreign bodies in the background of the eye. ARCH. OF OPHTH., xxiii., p. 172.

136. BROSE. Two cases of electrical flashing, followed by severe retinal irritation and intense eye pains. *Ibid.*, p. 124.

137. RIVERS. Injury to the eyes from a heavy charge of electricity. *Ibid.*, p. 31.

HIRSCHBERG (128) reports four successful extractions of a foreign body from the retina, and one in which the location of the foreign body could not be found, and removal was impossible. He replies to the many objections made to the method by those favoring the extraction with Haab's strong magnet, and speaks of suppuration in the wound and in the interior of the eye, and the loss and destruction of the vitreous.

In DEUTSCHMANN'S (130) case Hirschberg's electro-magnet did not remove the foreign body, but this was successfully accomplished with a larger apparatus.

HAAB (131) found by clinical observation and experiments on animals that neither the Ruhmkorff apparatus nor the horseshoe magnet is adapted for the extraction of iron splinters from the eye. He therefore had constructed a large magnet, with conical points, one of which is brought near the eye ; 6–8 ampères and 50–60 volts are usually employed. Any iron splinter weighing over 0.02 gr. is drawn forward behind the iris or into the anterior chamber. When the magnet is brought to the centre of the cornea the foreign body is not drawn through the lens, but around it. The instrument is also useful in diagnosis, as it produces pain, except when the foreign body present is imbedded in the retina.

Gerard's magnetometer, presented by GALLEMAERTS (132) to the French Ophthalmological Society, demonstrates the presence of and localizes iron splinters weighing a half milligramme.

SULZER.

HÜRZELER (134) found that 90 % of the foreign bodies seen in the Zurich Clinic were in the conjunctiva or cornea, 7 % in the lens, and 6 % in the vitreous. In 313 magnet operations collected from various sources, the foreign body was removed in 65 % of cases ; some vision was retained in 69 cases, preservation of the ball in 35, and complete loss of the eye in 99. Serviceable vision was rarely obtained, and detachment of the retina often followed later. In 18 cases in the Zurich Clinic the results were better, but in 5 cases the foreign body could not be removed by Hirschberg's method. Haab's stronger magnet, in the use of which the vitreous is not disturbed, is a decided advance. One should endeavor to have the foreign body follow the direction of its entry, and the operation should be done early.

KNAPP (135) reports four cases in which foreign bodies remained a long time in the fundus. When the eye is free from irritation it is well not to operate unless the case is seen early. When there is only one eye the treatment should always be expectant. In two such cases Knapp ordered rest in bed, and later operated on the traumatic cataract, obtaining good vision, which was permanent. GREEFF.

BROSE (136) adds two cases to the two previously reported of retinal irritation after a severe electric shock. The eyes could not be opened and were painful. The conjunctiva was injected, the pupils small, and the retinal vessels overfilled. The pain and photophobia passed off gradually. GREEFF.

RIVERS'S (137) patient received a strong electric shock that threw him down and burned the skin of the lids and face. The opaque epithelium of both corneæ was cast off in a few days and regenerated as completely transparent epithelium. A marked photophobia persisted, which the author believed not to be due to the irritation of the retina by the electricity, but to the blinding of the retina from the bright flash of light at the time the shock was received. GREEFF.

XXI.—OCULAR DISTURBANCES IN GENERAL AFFECTIONS.

138. VIALET. Considerations on the visual centre, with two new cases of cortical hemiopia with autopsies.

139. JOCQS. Bilateral homonymous hemiopia with preservation of central vision. *Soc. franç. d'opht.*, 1894.

140. TOMKINS. A case of sensory aphasia, accompanied by word deafness, word blindness, and agraphia. *Brit. Med. Jour.*, April, 1894, p. 907. (No post-mortem ; nothing new.)

141. PIC. Note on a case of tumor of the occipital lobe with hemiopia. *Rev. génér. d'opht.*, No. 4, 1894.

142. SELL. On chiasm-disease, with report of a case that recovered. *Inaug. Dissert.*, Leipsic, 1894.

143. MOXTER. On the oculo-pupillary symptoms with paralyses of the upper extremity. *Inaug. Dissert.*, Berlin, 1894.

144. DE BONO and DALTO. The eyes of epileptics. *Arch. di Ottalm.*, i., 7–9, p. 234 ; and *Arch. f. Augenheilk.*, xxix., p. 66.

145. BOREL. Traumatic hysteria of the eye. *Arch. f. Augenheilk.*, xxix., p. 66.

146. Wolffberg. Objective symptoms of neurasthenia. *Zehender's klin. Monatsbl.*, xxxii., p. 128.

147. Müller, P. On ocular affections with and following measles. *Inaug. Dissert.*, Berlin, 1894.

148. Dreschfeld. A case of acromegaly. *Brit. Med. Jour.*, 1894, p. 5.

149. Huth. A case of actinomycosis of the eye. *Centralbl. f. prakt. Augenheilk.*, xviii., p. 106.

150. Berger. The action of toxines on the lachrymal secretion. Pathogenesis of keratomalacia following infectious diseases. *Rev. génér. d'opht.*, 1894, No. 5.

151. Chiarini and Fortunati. A case of lepra with ocular symptoms. *Ann. di Ottalm.*, xxiii., 1–2, p. 95.

In a tabic patient, aged sixty, Vialet (138) found a left homonymous hemiopia. Four years before, the patient had had an attack of apoplexy without paralysis. Death from gangrene of the lower extremities. A large focus of degeneration was found between the calcarine fissure and the occipital convolution. In the second case the focus was still larger. Vialet holds to his previous view that the cortical visual centre has a greater extent than the immediate neighborhood of the calcarine fissure.

Sulzer.

A woman of fifty-eight had a right homonymous hemiopia four years previously. Two years later an apoplectic attack led to complete blindness. Five or six days later there began a progressive complete return of central vision. Both visual fields extended 10° from the fixation point in every direction. Jocqs (139) concludes from this that there are two central visual spheres, one for peripheric and one for central vision. In most cases both are involved together, but in some cases the centre for peripheric vision is alone involved. Sulzer.

In Pic's (141) case of glioma of the right occipital lobe there was transient homonymous hemiopia leading to complete blindness, at first in one eye and then in the other. Sulzer.

Sell's (142) case was probably one of inflammatory exudation about the chiasm with partial serous infiltration. Recovery took place. In 91 cases reported 7 were due to injuries, 25 to tumors, 6 to tubercular exudation, 4 to aneurisms, 1 to pressure in the third ventricle, 2 to periostitis, 4 to partial meningitis, 16 to gummous basilar meningitis, 1 to hemorrhage, 1 to multiple sclerosis, and 1 to hyperplasia in elephantiasis and diabetes. Syphilis was present in 20 per cent. of the cases.

According to MOXTER (143) the partial paralyses of the brachial plexus may be divided into those involving the upper roots (Erb's paralysis), and those involving the three lower roots. In the latter there are oculo-pupillary symptoms, consisting of myosis, drooping of the upper lid, and retraction of the ball. The symptoms depend upon paralysis of the sympathetic fibres which arise in the cilio-spinal centre of the cord and pass in the course of the cervical sympathetic and sympathetic meshwork of the internal carotid to the unstriped muscle of the ball and orbit. The location of the alteration may be in the communicating branches of the 1st and 2d dorsal nerves.

In DRESCHFELD'S (148) very carefully recorded case, a man aged thirty, there was no perception of light in the left eye. Right eye $V = \frac{6}{60}$. No hemianopsia or color blindness. Optic atrophy more advanced in left eye. WERNER.

HUTH (149) incised what was taken to be a cyst in the lower lid nasally from the canaliculus, and scooped out several bodies imbedded in granulation tissue which proved to be actinomyces.

MISCELLANEOUS NOTES.

REVIEWS OF BOOKS.

In preparation : **An [Excellent] Ophthalmoscopic Atlas,** 75 plates with text. By Dr. J. OELLER, Munich. Sold by subscription mk. 75 ($18.50), in regular book trade mk. 100. Samples of these plates were shown at the Congress of Edinburgh, and numerous subscriptions were taken. The execution is in oil, and the plates were handed round in detached copies, provided with a border of thick Bristol board, for convenient use in class teaching. The atlas promises to be the most useful of its kind, and no doubt will be very popular. The publisher, J. F. Bergmann, Wiesbaden, solicits subscriptions sent to him directly. The work is to appear in numbers, and will be complete in two years.

H. K.

A Clinical Manual of Diseases of the Eye, including a Sketch of its Anatomy. By D. B. ST. JOHN ROOSA, M.D., LL.D., Professor of Diseases of the Eye and Ear in the New York Post-graduate Medical School and Hospital, etc., etc. 8vo, 617 pages. Wm. Wood & Co., New York, 1894.

The well-known author, who has more than anybody contributed to the development of systematic post-graduate medical teaching in the United States, says in the preface to the above work that it had not been his aim to prepare a cyclopedic text-book of ophthalmology, but chiefly to offer to the profession, in a permanent and accessible form, some of the results, with their personal coloring, of his long experience in ophthalmic disease and therapeutics. Judged from this standpoint the book contains a good deal of matter that is interesting and instructive even to the adept, though he may not always share the author's convictions. As a systematic treatise, where the different departments should be presented with a just and impartial estimate of their importance and limitation, according to the principle of the *suum cuique*, the book is unsatisfactory. We may except from this adverse criticism the first 116 pages contributed by Dr. A. E. DAVIS, in which the student will find a practical, clear, and comprehensive description of the anatomy and physiology of the visual organ. Particularly good in the subsequent part is the description of the construction and use of the Javal-Schiötz ophthalmometer. This is the author's favorite instrument, which he praises at the expense of other objective methods of testing refraction, for instance retinoscopy. The author's attitude toward the advances of modern science will not be enhanced by facetious remarks as the following: "Those who are satisfied with the statement that morbid germs cause all diseases, will no doubt say that hordeolum results from local infection with an hordeolum bacillus." The patriotic author has a laudable desire to base his descriptions, wherever possible, on American authorities, with a benevolent leaning toward his hospital associates and other friends, yet passages like the following might just as well be omitted in future editions: "Dr. D. has used this improved ratchet in his office for about two months." "In a strict sense, as was pointed out by V. F., the cornea and the lens form the chief seats of errors of refraction." "In acquired myopia from spasm of accommodation retinoscopy is useless, as the antero-posterior diameter of the eyeball is increased by the action of the ciliary muscle upon the lens." "In nasal hemianopia the lesion is situated in front of the chiasm and in temporal hemianopia just behind the chiasm," when, in fact, lesions in front or behind the chiasm can produce temporal hemianopia only, as is evident from the figure on page 91 (taken from Fuchs's text-book), to which the author refers immediately before the above sentence. Curiously this surprising blunder occurs also in a recent work on nervous diseases (Dana), on which the author repeatedly draws. Such unimportant inaccuracies, however, which occur in every original work, do not mar the usefulness of the book and are amply compensated for by the author's elaborate presentation of the importance of hyperopia and astigmatism, and his successful warfare against the exaggerated claims of those who have devoted special attention to the disturbances of motility.

H. K.

Traité Élémentaire d'Ophtalmologie. By H. NIMIER and F. DESPAGNET. A well gotten up gr. oct. vol. of 952 pages, a colored plate, and 432 figures in the text. Paris, Felix Alcan, 1894. Price frs. 20.

In many respects this is an excellent work. Written in the clear, transparent style, so characteristic of the French, it has also the more substantial advantages of weightiness and originality of matter to recommend it. Many of the clinical descriptions are very good, and give evidence of being penned by practised observers. Particularly good in this respect are the sections on cataract, glaucoma, functional disorders, and optic-nerve diseases, the latter being especially well presented. The various affections of the choroid and retina are also well described and differentiated, although the symptomatology might be sketched more in detail, and the account of tuberculosis of the choroid seems inadequate. Diseases of the lids are well described, and those of the conjunctiva, cornea, and iris fairly so, barring the account given of trachoma, which is insufficient. The theory of refraction is treated with all the lucidity of the French when dealing with a subject of exact science. On the other hand, the account of ophthalmoscopy, skiascopy, and the other objective methods, although full enough, is rather confused and disjointed. Here, as well as in the section on muscles and elsewhere, the authors have made some very careless mistakes—the tables of diagnosis of muscular paralysis being a flagrant example.

The plan of the book is excellent, the anatomy, pathology, and surgery of each portion of the eye being taken up separately and in the proper order. The anatomical descriptions and the portions relating to physiology are clear and sufficient; the remarks on treatment are generally judicious.

The cuts are mostly old friends, and, we may add, are not reproduced in the highest style of art. They are, however numerous, and illustrate the subject-matter sufficiently well.

On the whole, the book may be recommended for study; and to the ophthalmic practitioner it will afford many useful hints, and furnish him with the results of not a little original observation. A. D.

Farbenlehre. By Prof. L. MAUTHNER. Being Part I. of the "Functional Examination," thoroughly revised. Pp. 167. Bergmann, Wiesbaden, 1894.

This book deals with the doctrine of color in so far as it concerns the physiologist and ophthalmologist. It treats first of the perception of color, and of the production of color by means of polarization, spectral decomposition, the rotating disc, and the like. The main portion of the book is then taken up with a consideration of the various forms of color blindness and the tests for their detection, including both the more complicated experiments of the physiological laboratory, and the simpler methods of the clinician. The book concludes with a discussion of the different theories of color perception as they are held to-day.

The author has personally verified the observations and experiments of previous workers in this field, and has given us an intelligent and critical exposition of the subject, couched in the attractive literary style which distinguishes his writings.

W. A. H.

Transactions of the Eighth International Ophthalmological Congress, held in Edinburgh, August, 1894, edited, with the assistance of Drs. Parent, Hess, and Fergus, by Geo. A Berry, General Secretary. The nicely gotten up, rather sparsely illustrated volume of 352 pages will be welcome to all ophthalmologists, to those that had the pleasure to attend the Congress as a welcome souvenir of days profitably and pleasurably spent in the beautiful Scotch city, and to those that could not attend, as interesting reading of what oculists of all parts of the world had to say on their experience, investigation, and thinking.

H. K.

A new French ophthalmological journal, **La Clinique Ophthalmologique**, published under the direction of Dr. R. JOCQS, late hospital assistant of Paris. Published monthly by the Société d'Éditions Scientifiques, 4 Rue Antoine-Dubois, Paris. Frs. 10, yearly.

The object of this periodical is to furnish a great variety of news, especially clinical observations, for all ophthalmic practitioners, by short communications, including those of the most modest kind. At the head of each number of thirty two quarto pages will be a more important original article. In the first number (January, 1895), this article is on "Anterior Sclero-Choroiditis," by Dr. Ch. Abadie, who recommends colchicine as a "veritable specific" for this affection. Then follow several clinical contributions, society reports, abstracts from literature, book notices, and the beginning of a text-book chapter (on conjunctivitis), printed on the cover, for the general practitioner. H. K.

The February number of 1895 of the *Annales d'Oculistique* which we just receive announces that from January of this year on, an **English Edition of the "Annales,"** an exact reproduction of the Paris Edition, will appear in New York, under the direction of Dr. GEO. T. STEVENS, 33 West 33d St., New York.

At the same time we receive the first (January) number of the English Edition, published by the Transatlantic Publishing Company, 63 Fifth Ave., New York. Annual subscription price of the English Edition $5. This is certainly an excellent extension of the oldest ophthalmic journal so vigorously continued by its present, ambitious, chief editor, E. Valùde, of Paris. The "Annalles" appear in monthly numbers of 80 pages each, half of which are devoted to original communications, the other half to reports of societies and current literature. There are no illlustrations. The style of the English Edition is very good. H. K.

APPOINTMENTS.

Prof. ISIDOR SCHNABEL, in Prague, has been appointed Prof. MAUTHNER'S successor, in Vienna. Mauthner died of heart failure a day after he had been appointed to succeed STELLWAG.

In the Jefferson Medical College of Philadelphia a **new full professorship,** has been created, namely, for **ophthalmology,** with Dr. WM. THOMSON as its first incumbent, having seat and voice in the council of the Faculty. At the same time Dr. H. F. HANSELL was appointed Clinical Professor of Ophthalmology, to hold this position together with Dr. GEO. E. DE SCHWEINITZ.

PRIZE ESSAY.

An essay, entitled "Toxic Amblyopias: their Pathology and Treatment," by Dr. G. E. DE SCHWEINITZ, Clinical Professor of Ophthalmology, Jefferson Medical College, has been awarded the Alvarenga Prize of the College of Physicians of Philadelphia.

Obituary.

On October 20, 1894, and in the fifty-fifth year of his life, Prof. LUDWIG MAUTHNER died of apoplexy at Vienna.

Born April 13, 1840, at Prague, he studied there and in Vienna, graduated at the latter university in 1861, and at the instance of that gifted but, at that time, misjudged and slighted Eduard von Jaeger, devoted himself mainly to ophthalmology. After a prolonged scientific course of travel, which carried him to Berlin with A. v. Graefe, and to London, Mauthner established himself, in 1864, in Vienna. In 1869 he obtained the regular professorship of ophthalmology in Innsbruck—a position, however, which, as he did not feel satisfied with the extent of his sphere of activity there, he gave up voluntarily, in order to return to Vienna as a privatdocent. A day before his death he attained that which for seventeen years had hovered before his eyes as the highest goal of his ambition—his nomination as regular professor of ophthalmology in the Vienna Medical Faculty, to succeed Stellwag von Carion.

As an author, Mauthner accomplished work much beyond the ordinary ; his writings are characterized by great acuteness and unexcelled industry and thoroughness. In 1868 appeared his *Textbook of Ophthalmoscopy*, the best work extant upon this department of ophthalmology. This was followed by his lectures upon the *Optical Errors of the Eye* (Vienna, 1876), a valuable contribution to the more recent ophthalmological literature. From 1876 to 1878 Mauthner was associate editor of the German edition of these ARCHIVES. In addition to his excellent monographs, embracing the whole range of ophthalmology, of which those upon the *Functional Examination of the Eye*, the *Sympathetic Affections*, the *Relations between Brain and Eye*, *Glaucoma*, and *Paralysis of the Ocular Muscles*, had already appeared, there was published a few weeks since his treatise on the *Theory of Colors*, which constituted the first part of the second and very much altered edition of the treatise on functional testing. All his writings are preëminently characterized by the extreme clearness of the presentation.

C. HORSTMANN.

MEETINGS OF SOCIETIES.

The French Ophthalmological Society will meet in Paris a l'Hôtel des Sociétés Savantes, opposite the École de Médecine, Monday, May 6, at 8.30 A.M. Titles of papers to be sent in on or before March 15th. Subject for general discussion : The Comparative Value of the Different Objective Methods of Optometry ; to be introduced by Dr. Parent. The same subject was chosen for general discussion in the ophthalmic section of the American Medical Association at its meeting in San Francisco last year.

The next meeting of the American Medical Association will be held at Baltimore, Md., from May 7 to 10, 1895. *Ophthalmological Section :* Pres. Edw. Jackson, Philadelphia ; Sec'y H. V. Wuerdemann, Milwaukee, Wis. *Laryngological and Otological Section :* Pres. J. F. Fulton, St. Paul, Minn.; Sec'y T. J. Gallaher, Pittsburgh, Pa.

Contents of the last numbers of the Archiv für Augenheilkunde (German edition of these ARCHIVES).

Vol. XXIX., Nos. 3 and 4, issued November, 1894.

A.—ORIGINAL PAPERS.

XII. R. DENIG. Bilateral Distortion of the Outer Commissure of the Lids Due to Congenital Contraction of the Platysma.

XIII. SCHWEIGGER. The Results of Squint Operations. (Translation in this number.)

XIV. MANZ. Opaque Nerve Fibres of the Retina. Plate I. and II.

XV. THEO. CHRISTEN. Three Cases of Congenital Coloboma of the Lid. Plate III.

XVI. C. MELLINGER. Subconjunctival Injections and their Therapeutic Value.

XVII. G. GUTMANN. Subconjunctival Injection.

XVIII. CRAMER and SCHULTZE. Pseudogliomas of the Retina. Plate IV.

XIX. L. WEISS. Action of Internal and External Recti in Increasing Divergence. Plates V.–VII.

XX. R. GREEF. Spider Cells in Optic Nerve and Retina. Plates VIII. and IX.

XXI. R. NIEDEN. Sympathetic Inflammation from Choroidal Sarcoma. (See his Report of the Edinburgh Congress, these ARCHIVES, year 1894, p. 468.)

XXII. VELHAGEN. Experiments on Optic Neurectomia in the Rabbit. Plates X. and XI.

XXIII. GUILLERY. Latent Disturbances of the Ocular Muscles in Tabes.

XXIV. H. Knapp. Tolerance of Foreign Bodies in the Background of the Eye. (See year 1894, p. 172 of these Archives.)

B.—REPORTS.

Eighth International Medical Congress. See last number of these Archives.

Systematic Report on Progress, First Quarter of 1894. See last number of these Archives.

Vol. XXX., No. 1, issued December, 1894.

A.—ORIGINAL PAPERS.

I. H. Knapp. Glaucoma after Discission of Cataract; its Cure. (Translation of an article published in these Archives in 1892, p. 293, with remarks and additional cases.)

II. L. Bach. Experimental Nystagmus in Healthy Persons and Deaf-Mutes.

III. Kalt. Corneal Suture in Extraction of Cataract. (Published in October number, 1894, of these Archives, p. 421.)

IV. L. Weiss and W. Ottinger. Congenital Malformations of the Eye.

V. R. Gruber. Silver Impregnation of Corneal Tissue.

VI. Hillemanns. Injuries of Eye.

VII. Translations and Reports of the Original Papers of Nos. 1 and 2, 1894 of these Archives. By R. Greef.

B.—SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Second Quarter of 1894. (Translation in this number.)

VOL. XXIV. No. 2

ARCHIVES OF OPHTHALMOLOGY.

ON THE PROBABLE HEMORRHAGIC ORIGIN OF THE STRIATED AFFECTIONS OF THE RETINA.[1]

With reports of cases from the practice of Dr. H. KNAPP and the author.

BY DR. WARD A. HOLDEN, NEW YORK.

(*With two illustrations.*)

ONE of the weak points in ophthalmoscopic diagnosis is the frequency with which we are unable to distinguish conditions that are the result of hemorrhage, from conditions that are due to primary inflammation; although the more careful study of retinal affections in late years has taught us that many conditions supposed to be inflammatory represent in fact the metamorphosis of hemorrhage.

Of the three distinct varieties of new-formed striations in the retina, two are known to be the result of hemorrhage, while the genesis of the third is still unknown.

A case to which I wish to ask your attention to-night combines in a measure the characteristics of all these varieties, and in it the development of striations from a fresh hemorrhage was clearly followed. This, together with the analogous appearance of many cases of the unexplained variety of retinal striations, leads me to suggest that this variety also may be the result of hemorrhage.

The first variety is the condition in which on or just beneath the surface of the retina, and mostly overlying the retinal vessels, are strands of bluish-white glistening membrane. This has frequently been seen to follow traumatic or idiopathic hemorrhage, and Manz has called it retinitis proliferans. This designation seems proper since it has recently been shown that there is a proliferation of Müller's fibres and a deposition among them of new connective tissue.

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[1] Read before the Ophthalmological Section of the New York Academy of Medicine, November 19, 1894.

The second variety is the very rare condition in which a number of narrow branched brown stripes lying *beneath* the vessels of the retina run from the periphery of the fundus toward the disc. Plange,[1] who described the first case reported, saw these stripes develop in the course of repeated hemorrhages. Knapp[2] reported a case calling the affection angioid streaks in the retina, and Stephenson[3] reported another in which many of the brown stripes were bordered on one side only by a broader white stripe.

The third variety is the rather rare condition in which lying beneath the retinal vessels are narrow yellowish-white striæ, sometimes branched, and often bordered on either side by a narrow line of pigment; the striæ extending in general direction from the periphery toward the disc. This affection was pictured by Jaeger and was later called by Nagel retinitis striata. Its etiology is unknown.

All of these varieties of striations are permanent conditions, and are not easily confounded with the inflammatory stripes in *syphilitic* retinitis, which run in the direction of the vessels and may disappear under specific treatment.

The case to be reported and most of those to be briefly mentioned later, have been seen in the private practice of Dr. Knapp, and through his kindness I am permitted to make use of them.

Miss M., aged forty-three, was first seen on July 5, 1894, when she complained of gradual failure of sight for a year. The patient was of slender build, was nervous and anæmic, and had frequent attacks of giddiness. She had had no serious illness of recent years, and there were no discoverable organic affections, and no menstrual disturbance. The acuteness of vision was R. $\frac{20}{200}$; L. $\frac{20}{100}$, not improved with glasses. The limits of the fields for white and colors were normal. The media were clear. The optic discs and the retinal vessels presented no peculiarities. About the disc in each eye was an irregularly outlined zone in which the retina was somewhat opaque, obscuring the red reflex from the choroid. In each macular region there was disturbance of pigmentation with scattered small hemorrhages, dark spots, and superficial bluish-white membranous patches.

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[1] Arch. of Ophth., xxi., 2, p. 283, 1892.

[2] *Ibid.*, p. 299.

[3] *Trans. Ophth. Soc., Un. K.*, xii., 1892.

Radiating from the pale zone about the disc, in each eye, was a system of branched striations of dark-brown color, having each about the width of a vein on the optic disc, and lying beneath the retinal vessels. In the left eye this system of angioid streaks closely resembled those described in previous cases, and does not particularly interest us. In the right eye many of these angioid streaks were bordered on one side only, by a light streak double the width of the dark. In the temporal periphery of the right fundus was a patch of opacity in the retina, from which narrow processes branched out, each bordered with a delicate line of pigment. Temporally and below the disc a large hemorrhage of

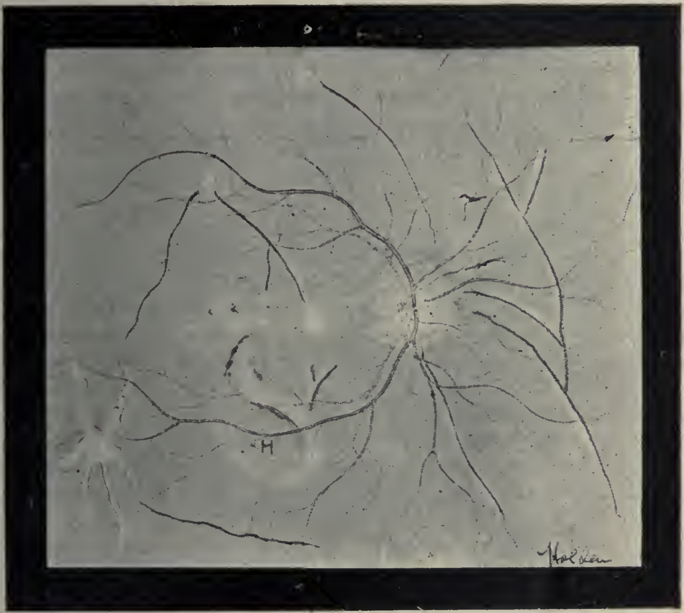

FIG. I.

Pigmented Angioid Streaks in the retina. With a hemorrhage, "× H" in the process of absorption and a new branched streak forming above it. *Erect image.*

bright red color, irregularly branched in form though sharply outlined, lay beneath a retinal vein. When seen three weeks later the hemorrhage had assumed a more regularly elliptical form, and the surrounding retina had become milky. The hemorrhage then began to grow paler below, while the superior margin remained bright red. As the hemorrhage became further absorbed a narrow process of red color pushed upward from its superior margin and gradually extended a distance of two disc-diameters in a curved streak, but slightly broader than a vein on the disc. It still was red when the patient was last seen, four months after the first visit.

While the hemorrhage was undergoing absorption from below, there appeared just above it a short streak of dark-brown color, bordered on one side by a broader light streak. This double streak branched like a Y, as is shown in the drawing, and one branch gradually extended upward until it became two disc-diameters in length, while the shorter branch became partially absorbed. This new streak was similar in color and conformation to the older ones.

When examined in the erect image all the dark streaks were seen to consist of a stippling of brown. The lighter bordering streaks did not elevate the vessels of the retina, and seemed to be simply a slight opacity of that membrane.

The patient was kept under observation four months and then left the city permanently. Preparations of iron were given and later iodide of potassium, and no fresh hemorrhages occurred. No change was observed in the condition of the fundus except in and near the fresh hemorrhages.

The large hemorrhage became absorbed from below, the blood collecting at its sharp upper margin, the narrow streak of blood extended upward from it, and the new double streak developed just above it, extending in a general direction upward toward the disc, as if there were currents in the retina running centripetally.

Most of the older streaks ran from the periphery toward the centre, as in the cases previously reported, and were gradually lost in the neighborhood of the disc. Some of these older pigmented streaks arose in the periphery from whitish membranous patches, like those found in retinitis proliferans, and one of these patches gave origin to two streaks. Thus these old striæ would seem to have developed like the new one, apparently by the elements of a peripheric hemorrhage being carried toward the disc in a path lying in the deep layers of the retina.

In studying cases of so-called retinitis striata, one is struck not only by the similarity in course and conformation between the white stripes seen in this affection, and the pigmented angioid streaks, but also by the presence of patches of opacity or of pigmentation of the retina, suggestive of previous hemorrhages, from which many of the white striæ arise.

The cases of retinitis striata that I have myself examined are as follows:

Case 1.—Miss M., aged twenty-five, noticed a blurring of vision three weeks before the examination. Two days previously she found that the sight of the right eye was very poor, and examination of the field of vision revealed a central scotoma. In the inferior portion of the fundus were long stripes, and a network of glistening whitish bands lying beneath and elevating the vessels of the retina. One of these stripes ran through the macula and ended in a line of pigment. Near the macula were small pigmented patches, and to the nasal side of the disc were larger patches of rarefaction and pigmentation. The retinal veins were enlarged.

Case 2.—A boy, aged twenty, following an acute nephritis, had swelling of the discs with white patches in the region of the macula, and hemorrhages scattered through the fundus. Six months later the changes in the disc and about the macula had disappeared, but some of the inferior retinal vessels were bordered with white lines, and in the inferior periphery of the fundus in both eyes were long white stripes ending in patches of pigment.

Case 3.—Miss F., aged thirty-seven, always myopic, noticed a sudden blurring of vision six years previously, which cleared again. Later there had been a gradual failure of sight, and at the examination the patient was only able to perceive the motion of the hand in the lower outer portion of the field. In the upper portion of the fundus were three narrow yellowish-white lines bordered by black, lying beneath the retinal vessels without elevating them, radiating from the neighborhood of the disc and ending peripherically in patches of slight opacity of the retina.

In the lower portion of the fundus was a stippled black stripe running horizontally, and there was increased pigmentation of the surrounding retina. The condition in the other eye was similar, but less extensive.

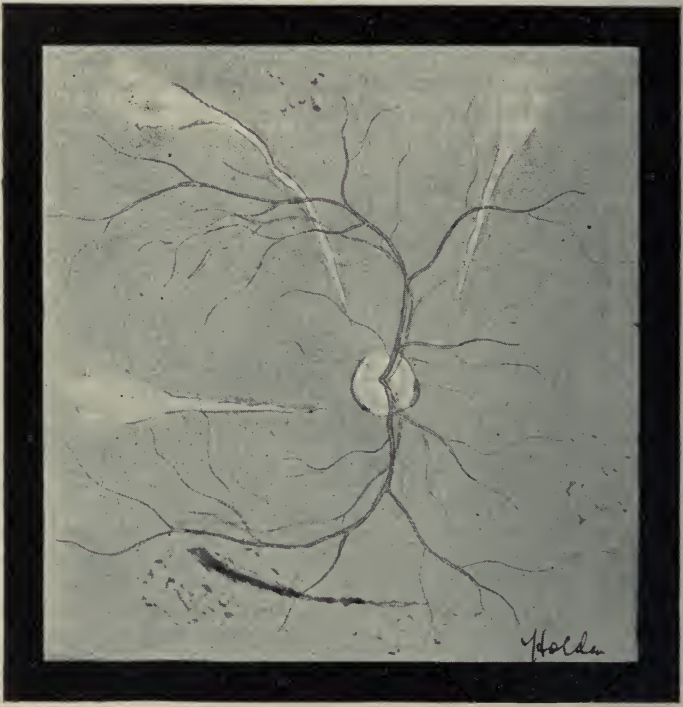

FIG. 2.—RETINITIS STRIATA.

Stripes of rarefaction and one of pigmentation.

In one of the cases pictured in Jaeger's atlas, two of the white stripes originate in dark-brown patches in the periphery, and run toward the disc.

Finally, not to multiply cases further, there is a drawing by Caspar in the Helmholtz Festschrift, in which there is a network of white stripes with black margins beneath the vessels of the retina, and near the disc several long branched hemorrhagic stripes, differing from angioid streaks only in their bright-red color. In looking at these, it would seem natural that there should have been suggested to the observer the probable hemorrhagic origin of the older white stripes, though Caspar considered the affection choroidal, with possibly some involvement of the retina.

Onesi,[1] writing in 1890, gave abstracts of nine cases of retinitis striata from literature, and in his general description of the affection says: "The light stripes lie beneath the retinal vessels and in front of the pigment epithelium, the choroid not being involved in uncomplicated cases. The pigment may be in the form of masses, or in granules bordering the light stripes. The stripes, often branching, extend from the periphery and gradually fade away near the disc, where the retina has sometimes lost its transparency. The stripes are broader in the periphery, and often end in gray or pigmented patches, or in shallow detachments of the retina. The stripes may or may not elevate the retinal vessels. The macular region may show light spots, white or red stripes, black patches, or rarefaction."

Thus the two affections, angioid streaks and retinitis striata present a remarkable similarity, differing mainly in the color of the stripes, and we know that the ultimate result of a retinal hemorrhage may be a patch of light color or one of pigmentation. In both these affections there are found changes that are known to be due to hemorrhage, while in both, changes that are known to be due to inflammation are for the most part wanting.

It would therefore seem warrantable to assume as a hypothesis, to be verified by future observation, that the affection called retinitis striata, like that called angioid streaks, arises through the elements of peripheric hemorrhages being diffused in a linear manner in the deep layers of the retina and undergoing various sorts of metamorphosis.

While it is probable that the diffusion may take place, in part at least, through previously existing lymph-channels, our knowledge of the lymph currents in the retina is still too indefinite to throw any light on the subject.

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[1] *Mittheilungen aus der Oph. Klinik in Tübingen*, 1890.

THE LENS-STAR FIGURE OF MAN AND THE VERTEBRATES.

By PERCY FRIDENBERG, M.D.

(*With 21 figs. on plate.*)

THE structure of the crystalline lens has been the object of so frequent scientific study, that it would seem to offer, at the best, an unpromising field for histological investigation. However, in spite of our accurate knowledge of most structural details of the crystalline body, we are still insufficiently informed as to the composition and proportions of an interesting and possibly important part of the lens, the inter-fibrillary cement or lens-star substance.

Since the early publications of Young, Werneck, Bowman, and Hannover, which were principally devoted to a study of the course of the lens-fibres, little has become known as to the form and composition of the lens-star figure. Nunneley first described the lens-star of vertebrates, and showed that the tri-branch was the type of this order. His view was supported by Huschke, and has been accepted by all later writers. In the human embryo, and at birth, the lens shows no change from this typical form; the three-pointed star of the vertebrates appears in its original simplicity. With the development of the organism and the increased bulk of the lens, however, a change takes place in the arrangement and number of the star-points. In a former publication[1] I reported the results of an investigation on this subject, in the course of which I was able to study the details of the lens-star during life in a large number of normal human eyes.

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[1] *Ueber die Sternfigur der Krystall-Linse*, I.-D., Strassburg, 1891.

My observations convinced me that the schematic representations familiar to us from perusal of the anatomical text-books and monographs, and generally accepted as correct, are not reliable, and that these drawings differ quite materially from the form of the lens-star as actually seen *intra vitam.* The theory that the change in form of the lens-star with the development of the individual would become evident as an increase in the number of its rays alone, led to the acceptance as typical of a mathematically subdivided star-figure of six, nine, or twelve rays.

Although this presumption had no basis in fact, and was unsupported by actual observation, these geometrically accurate figures were copied with unquestioning faith by one writer after another, and we find them unchanged in the works of Werneck, Huschke, Fr. Arnold, and others (*vide* Plate p. 159, Figs. 1–5).

The importance of an accurate knowledge of the true form of the lens-star warrants a brief recapitulation of the results of my investigation. The examination was made with the corneal loupe of Zehender-Westien, and the eye so placed that the anterior part of the lens was illuminated obliquely. The rays from the source of light were totally reflected from the anterior surface of the lens, which then presented a bright slate-gray reflex, allowing the details of its structure to be studied with ease. I found, first, a decided variety in the number and arrangement of the star-rays. The regularity of form which I had expected was conspicuously absent. In only one or two cases was I able to find a three-pointed star. Of one hundred lenses about one fifth showed four rays, two fifths presented a five-pointed star, one fifth a figure with six rays, the rest (seven cases) a seven-pointed star. I found no figures with more than seven primary rays, although secondary lines could be counted up to the limit of visibility, making the total of rays twenty or more. The figure at the pole of the lens is rarely that of a true star, as the angles between adjacent rays are usually unequal; besides this, the rays do not issue from the pole or from a point which corresponds to it approximately, but start, usually, from a sutural line running through the pole.

This line is in most cases vertical, but not at all infrequently oblique. Rays proceeding from the opposite ends of such a line produce a figure which appears like an inverted and an upright embryonal tri-branch, in which the vertical rays have joined at the pole.

The division of the rays was quite unlike that usually represented. The difference between the lens-star of the adult and that of the embryo does not consist alone, or even principally, in a dichotomous division and multiplication of primary rays. Dichotomous division is by no means the rule, as a ray may divide into two, three, or more secondary lines, while its course to the periphery is free from divisions on the other side. At times the division of rays is dendritic in character, and resembles somewhat the arrangement of retinal vessels (Laqueur), as the subdivision of rays is most active in the portions of the lens above or below the pole, and the lateral quadrants are less abundantly supplied (Figs. 6–9). An examination of seventy-seven lenses to determine the relative number of rays in the different quadrants of the lens, showed that on an average there were in the upper quadrant two or three, in the lower quadrant three or four, primary rays, with one or two in the nasal quadrant and one ray in the temporal.

Although the quantitative relations of the rays varied somewhat, it was almost invariably found that the number of rays became successively less in advancing from the inferior to the superior quadrant, and then from the nasal to the temporal. The relative frequency of the rays in the upper and lower quadrants varied within certain limits, so that the proportion became, at times, 4:3, 3:2, 5:4, in favor of the former. The relative paucity of rays in the temporal quadrant was constant and quite striking, the total number of rays here being in the proportion of 2:3 in the nasal, of 1:3 in the superior, of almost 1:4 in the inferior quadrant, and as 1:13 in relation to the total number of rays in the three other quadrants.

In regard to the lens-star of mammals I was able to find only sparse observations, and it is the object of this paper to report the result of a study of its structure in the lens of domestic animals. I have limited myself to lenses of the ox, horse, sheep, pig, dog, cat, mouse, rabbit, deer, and of several varieties of fishes, and in one or two cases have been able to compare the star seen *intra vitam* with the picture presented after staining the extracted lens. The following methods were employed to fix and stain the figures. An aqueous solution of Prussian blue was injected peripherally beneath the lens-capsule, and the excess of stain washed away in distilled water, leaving the star-figure outlined in deep blue.

This procedure was employed in the case of larger animals. The smaller lenses seemed too delicate to allow them to be subjected with impunity to this mechanical method. In this series I injected a solution of ferrocyanide of potassium into the anterior chamber of the living animal, and after death subjected the lens in its capsule to the action of ferric chloride in aqueous solution. The results of this method were not uniformly satisfactory, as the resulting blue stain sometimes became so intense as to obscure the details of the lens surface.

The most satisfactory reagent was found to be nitrate of silver in the strength of 1:500–1:1000. Robinski,[1] who claims that the lens-star is invariably an artificial product due to the irregular shrinking and cleavage of the lens surface after hardening in Müller's fluid, alcohol, and similar reagents, considers the silver solution free from all objection on this score, as it has no hardening or macerating action on the albumen of tissues. On this point he says: "I must call attention to the fact that imbibition takes place when the lens is immersed in hardening fluids. As the superficial layers of this body contain a greater percentage of water than the deeper (Kölliker) there is more absorption of fluid in the dry nuclear layers than in the watery cortex; the nucleus itself imbibes most freely, and exerts a bursting strain on the layers enveloping it. This is followed by a fissuring of the surface of the lens, with the production of so-called star-figures."

In his investigations, Robinski used nitrate of silver in solution (1:800), the fresh lens being immersed in this fluid for from five to fifteen minutes. He claims that no trace of a lens-star is seen in lenses treated in this way, which is the only process free from the objections raised against hardening fluids, as it leaves the surface of the lens unchanged. The silver in weak solution has no action on the lens fibres, but makes their contour visible, as a precipitation of metallic silver takes place along the lines of contact in the form of minute brownish-black granules. At first I intended to use this method for the study of the lens fibres, as it had been expressly stated by the above mentioned observer that he had found no trace of a lens-star. I immersed fresh lenses, in the capsule, in a weak solution of silver nitrate, and within five or ten minutes could see clearly the outlines of the lens-star figure, which gradually grew more and more distinct as the precipitation of silver progressed. In each case a drawing of the figure was made, and the lens left to the action of the silver for twenty-four hours.

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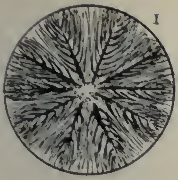
[1] *Med. Centralblatt*, 1888.

Meanwhile the entire surface of the lens took on a rich chocolate color, the rays of the star-figure having become intensely black and offering a strong contrast.

There were absolutely no fissures or even the slightest break to be discovered on the smooth, highly reflecting surface of the lens. Microscopic examination with low powers also showed the surface to be perfectly intact, so that we may well assume that there has been no imbibition or manifestation of a "bursting strain," and that there is no doubt of the star-figure being a natural structure which can be made visible by the simplest methods. The results of this staining method are equally satisfactory for small or large lenses. The lens-star of mammalians corresponds in general to that of man. In the young the tri-brachial type of the vertebrates is seen unmodified (Fig. 10), while in fully developed animals the rays are more numerous (Figs. 12, 15, 18), somewhat irregular in distribution, and may arise, as in the human lens, from a sutural line instead of meeting in a common pole (Figs. 12, 15, 19).

The calf lens (Fig. 10) shows small lateral twigs running from the primary rays of the figure, which resembles the condition seen in man. Ox lenses (Figs. 11, 12) show a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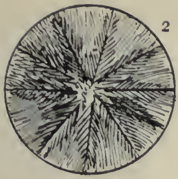
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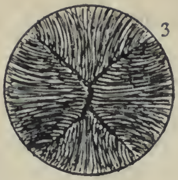
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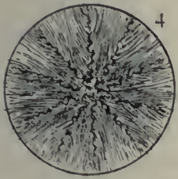
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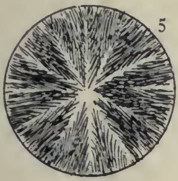
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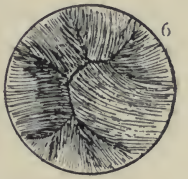
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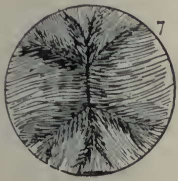
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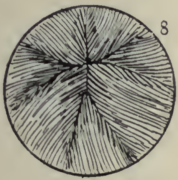
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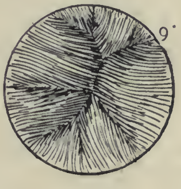
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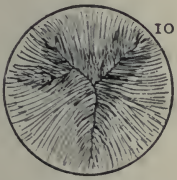
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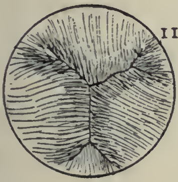
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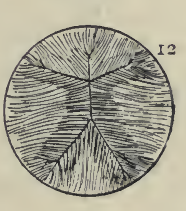
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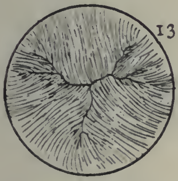
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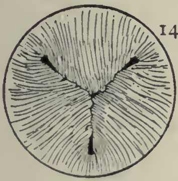
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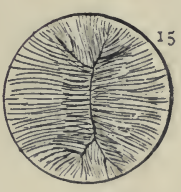
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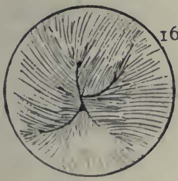
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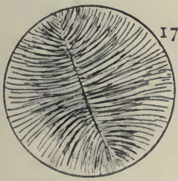
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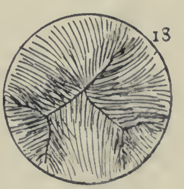
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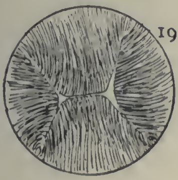
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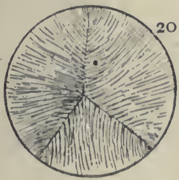
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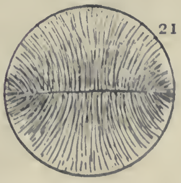
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P. FRIDENBERG, del.

more complicated star than that of the calf, although the three-point figure is still evident. The irregularity in distribution of primary rays, with the increase in number of the branches, and the relative prominence of lateral twigs are particularly noticeable. The lens of the mouse (Fig. 16) shows a three- or four-rayed star with curved branches presenting their convexity to the pole. The lens of the rabbit (Fig. 17) differs from the others examined in presenting no star proper. This figure is replaced by a sutural line running through the pole, from above usually to the lower outer quadrant. A similar line takes the place of a star-figure in the lens of fishes (Fig. 21).

EXPLANATION OF THE DRAWINGS.

Figs. 1–5. Schematic representation of a theoretically constructed lens-star.

Fig. 1. After Huschke, Edw. (Meckel's *Archiv*, 1832, Fig. V.).

Fig. 2. After Werneck (Ammon's *Zeitschrift*, vol. iv. and v., Fig. V.).

Fig. 3. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 17).

Fig. 4. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 18).

Fig. 5. After Arnold, Fr. (*Tabulæ Anatom.*, Fascic. ii., 1851, Fig. 19).

Figs. 6–9. Star-figures of the human lens as seen with the corneal loupe, showing the origin of secondary rays from a sutural line and their distribution in the different quadrants.

Fig. 6. Left lens. Adult (thirty-three years).

Fig. 7. Left lens (sixteen years). Frequent form.

Fig. 8. Left lens (thirty-two years). " "

Fig. 9. Right lens (twenty-eight years).

Figs 10–21. Star-figures of the lens of animals treated with nitrate of silver, 1:500.

Fig. 10.	Lens of calf.	Magnified	1.5x.
Fig. 11.	Lens of ox,	"	1.25x.
Fig. 12.	Lens of ox.	"	1.25x.
Fig. 13.	Lens of sheep.	"	1.75x.
Fig. 14.	Lens of pig.	"	2x.
Fig. 15.	Lens of cat.	"	2x.
Fig. 16.	Lens of mouse.	"	10x.
Fig. 17.	Lens of rabbit.	"	2.5x.
Fig. 18.	Lens of horse.	"	1.25x.
Fig. 19.	Lens of buffalo.	"	1.25x.
Fig. 20.	Lens of deer.	"	1.5x.
Fig. 21.	Lens of salmon.	"	3x.

A TUMOR OF THE CILIARY BODY; WITH REMARKS.

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DOCENT IN OPHTHALMOLOGY AT PRAGUE, AUSTRIA.

Translated by Dr. Ward A. Holden.

(*With Plate VI., Vol. XXVIII., Germ. Ed.*)

M. W., a man of sixty-six was first seen early in September, 1893, when he complained of having had a dull feeling of pressure in the right eye for four months. Vision was reduced and he saw halos. There was + T ½; and in the lower outer quadrant of the chamber angle, a gray stripe of foreign matter, 0.5 *mm* wide, separated the periphery of the iris from the ciliary body and projected forward above the level of the iris. The upper inner half of the posterior capsule of the lens was diffusely opaque. Behind the lens and corresponding in position to the foreign substance at the periphery of the iris was a rounded blackish tumor the size of a pea. Fundus normal; V = ⅓.

Sept. 23d.—A brown point was seen under the conjunctiva 2 *mm* from the limbus, in the middle of the lower outer quadrant of the sclera, corresponding in position to the point of perforation of an anterior ciliary artery. The mass at the periphery of the iris was broader, and the pupil had assumed a kidney form, due to muscular paralysis in the affected portion of the iris.

Dec. 28th.—There was a more marked iridodialysis caused by the tumor, and the pupil was displaced upward and inward. The projecting tumor mass, 2 *mm* broad by 6 *mm* long, was of white color. The lens was more opaque; the tumor mass touching it was larger, more rounded, and of dark-gray color. The sclera nodule was larger.

The patient had an attack of iritis., relieved by atropine. The lens became more cloudy, the vision was further reduced, and the scleral nodule increased in size. The ball was enucleated and hardened in Müller's fluid.

Macroscopically there is a loaf-shaped tumor in the lower anterior ciliary region, 9.5 *mm* long and 4.5 *mm* broad—Fig. 1. Posteriorly it passes gradually over into the normal choroid, anteriorly it is broader and pushes the iris insertion 3 *mm* inward and projects into the anterior chamber. The tumor is slightly mottled with pigment. The lens is displaced, its margin adjacent to the tumor being flattened, and the capsule being adherent to the tumor.

Macroscopically the tumor is seen to be composed of long and short spindle-cells, running for the most part meridianally, but some bundles having an equatorial course, and a few running in the direction of the radiating portion of the ciliary muscle. The nuclei are mostly spindle-shaped, but some are rod-shaped or oval—Fig. 2. In transverse section the nuclei are mostly angular, and not round. The cells form compact bundles of different length and thickness, which divide in various ways and form a meshwork. The groups of bundles are separated by connective-tissue septa, but the septa are rarely found between individual bundles. Toward the choroid there is a more marked pigmented reticulum giving the tumor an alveolar appearance. In the periphery of the tumor the cells as well as the nuclei have an oval form. The older cells in the substance of the tumor are for the most part spindle-shaped, and many of these long spindle-cells resemble, in longitudinal section, unstriped muscle-cells. These cells lie mostly isolated in the neighborhood of blood-vessels. Some cell-bundles that are cut transversely also show a similarity to leiomyoma in the multiangular contour of the cells and in their grouping into bundles—Fig. 2.

Posteriorly the tumor is covered by the pigment epithelium and the pars ciliaris retinæ, but anteriorly it breaks through these. The tumor contains many blood-vessels. The ciliary processes are not recognizable except in a small portion anteriorly. The lens capsule near the equator has been detached and is adherent to the tumor—Fig. 1. The epithelium here is replaced by a layer of fibrous tissue several cells deep.

The iris appears as a continuation of the anterior portion of the tumor, the latter being intercalated between the iris and choroid. The tumor has invaded the tissue of the iris, however, and both the pigmented and unpigmented cells have proliferated and apparently form sarcoma cells.

The episcleral nodule consists of long spindle-shaped cells with rod-shaped nuclei, in thin layers between the scleral lamellæ, strongly resembling ciliary muscle. Pigmented cells could be followed from the primary tumor to the episcleral nodule along the anterior ciliary vessels.

In its clinical and macroscopic aspects this tumor does not differ from the thirty cases in the literature (Mules, *Trans. Ophth. Soc. Un. K.*, 1888), all being loaf-shaped or hemispherical, springing from a particular portion of the ciliary body, and soon leading to iridodialysis, opacity and dislocation of the lens, and later to the formation of episcleral nodules. They are mostly composed of long spindle-cells in interlacing bundles, and are irregularly mottled with pigment.

The similarity of all these primary tumors of the ciliary body makes it seem probable that they all belong to a single tumor-group.

In only six cases were variations of this tumor-structure found, the spindle-cells, all or in part, being considered unstriped muscle-cells, and the terms myoma and myosarcoma of the ciliary body being used to designate the growths.

The more one works in pathological histology, the more one becomes convinced of the difficulty in a given case of distinguishing the pathological formation of unstriped muscle-cells from spindle sarcoma-cells and from young fibre-cells of fibrous tissue. Virchow (*Die Krankhaften Geschwülste*, vol. ii., p. 199) says that it is particularly difficult to distinguish spindle sarcoma-cells from unstriped muscle-fibres, and to separate certain varieties of myoma from spindle-celled sarcoma. It is easy to recognize the unstriped muscle-cells in the vessel-walls, uterus, and ciliary body in normal conditions, but it is almost impossible to distinguish isolated normal muscle-cells from isolated spindle sarcoma-cells, and the difficulty is increased in pathological cases.

In myoma one might think that the manner of arrangement of the cell-bundles and the numerous delicate connective-tissue septa that are so well seen in normal and hypertrophied uterus tissue and also in the ciliary body might be of assistance in making a differential diagnosis, but these conditions are not constant. The tissue from which the tumor arises and the wavy or even serpentine form of the cell-nuclei are points of some value in diagnosis.

After these general remarks based upon the study of unstriped muscle in normal and pathological conditions, of myomata and fibromyomata of the uterus, and of various forms of spindle-celled sarcoma, we shall examine the six cases of spindle-celled tumor of the ciliary body reported as myomata and myosarcomata, in order to see whether unstriped muscle-cells were really present.

The first case is that of Iwanoff-De Wecker, in the year 1867, which was first described as a pure myoma and later as a myosarcoma, one regarding it as exclusively muscular, the other as principally muscular. " The entire outer portion of the tumor toward the sclera is composed of spindle-cells with rod-shaped nuclei. In the middle portion the muscle-fibres run longitudinally. Among the muscle-fibres are numerous connective-tissue cells. Toward the vitreous, spindle-cells and branched connective-tissue cells predominate, making up nearly the entire inner portion of the tumor." No drawings were given.

In Salomon's case (*Trans. Ophth. Soc. Un. K.*, vol. ii., p. 263, 1882) Priestley Smith examined the tumor and stated that it had the microscopic character of a myoma or myofibroma, consisting of spindle-cells mostly arranged in the form of reticular fibrillæ and running parallel, presenting a great similarity to the tissue of the normal ciliary muscle. A committee, composed of Brailey, Mackenzie, and Nettleship, made the following report on the case: " The tumor consists of fine cells with rod-shaped nuclei, which cannot be distinguished from the cells of the ciliary muscle or the cells of a 'fibroma' (myoma) of the uterus, with sections of which it was compared. The arrangement of the cell-fibres in long bundles, which take principally an antero-posterior direction, but also run obliquely, is entirely similar to the relations of the normal ciliary muscle." No microscopic drawing is given.

The argument that a spindle-celled tissue with parallel fibres resembles the normal ciliary muscle, and that its spindle-cells resemble those of a fibroma and a myoma of the uterus, is not sufficient to convince me that the tissue was myomatous. For so many varieties of spindle-celled sarcoma tissue may resemble ciliary muscle, and it is the less convincing when the same tissue resembles uterine fibroma, fibromyoma, and myoma, tumors composed of connective-tissue-fibre cells that differ considerably from one another. And incidentally it may be remarked that Billroth even in 1880 wrote (*Die allgem. chirurg. Pathol. u. Therapie*, Billroth-Winiwarter, ix. edit., p. 718) of the fibro-myomata of the uterus, that it would be more natural to class these tumors with the sarcomata.

Mules's case (*l. c.*, p. 66) of myosarcoma of the ciliary body, reported in 1888, was examined by Brailey, who said: "The neoplasm consists of elongated spindle-cells with slender nuclei, the cells forming a network of bundles with occasional large spaces between them; toward the surface of the tumor, particularly the inner, the cellular tissue is denser and the nuclei more numerous, and the reticular arrangement can hardly be made out, and the cells here can scarcely be considered spindle-cells. The reticular appearance is similar to that of a cavernous tumor, but I believe that the tumor is allied to unstriped muscular tissue, and that it has taken its origin from the muscular tissue of the ciliary body, the structure of which it resembles." Dreschfeld and Harris regarded the tumor as sarcomatous, and Robinson thought it a myosarcoma. No drawing was given.

The only spindle-celled tumor described as a pure myoma was that of Lagrange in 1889 (*Arch. d'opht.*, ix., p. 484). In meridional sections almost all the muscle-elements are cut transversely. Between the bundles which these fibres form are groups of dark-stained embryonic cells. In sections demonstrated by Ferré, quantities of young embryonic cells in rapid growth, identical with sarcoma cells, were found. This, however, seemed to be accidental, and Lagrange found them in none of his sections. Besides small islands of connective-tissue cells the sections contain only muscle-fibres and blood-vessels. The muscle-fibres form bundles separated by broad spaces. Lagrange believed that the meridional fibres of the ciliary muscle were preserved, and the radial portion of the fibres also did not take part in the pathological process. All the neoplastic fibres are situated internal to the posterior portion of the meridional fibres and in the anterior portion of the choroid. They represent an abnormally large radiating muscle which from Müller's muscle is distributed through the entire thickness of the ciliary muscle. Two drawings are said by Lagrange to represent unsatisfactorily a pure myoma, with which I quite agree.

Deutschmann says of his case of myosarcoma of the ciliary body in 1890 (*Beiträge zur Augenheilk.*, i., p. 72) that the tumor consisted of tracts of cells regularly arranged in a longitudinal direction, many of the spindle-cells being undoubtedly sarcoma cells, while many other tracts of cells were identical with unstriped muscle. No microscopic drawing is given.

Lange (*Graefe's Archiv*, xxxvi., p. 247) describes his case of myosarcoma of the ciliary muscle in 1890 as follows: "The tumor consists of delicate spindle-cells with rod-shaped nuclei. Most of these cells are arranged in meridional bundles. In the central portion are many bundles cut transversely, the cells of which then appear round or angular with small round nuclei. These parts appear alveolar. The nuclei are slightly clubbed at the ends, and the cells are distinctly larger than the unstriped muscle-cells of the normal part of the ciliary muscle." Lange says further: "On account of the great difficulty in distinguishing unstriped muscle-fibres from other spindle-formed connective-tissue cells it was not easy to make the diagnosis here. After careful study I came to the conclusion that my case was one con-

Fig. 1.

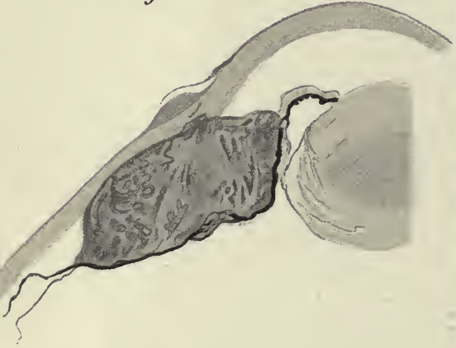


Fig. 2.

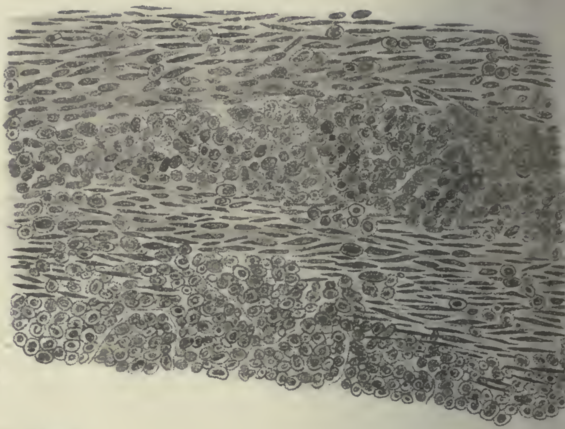
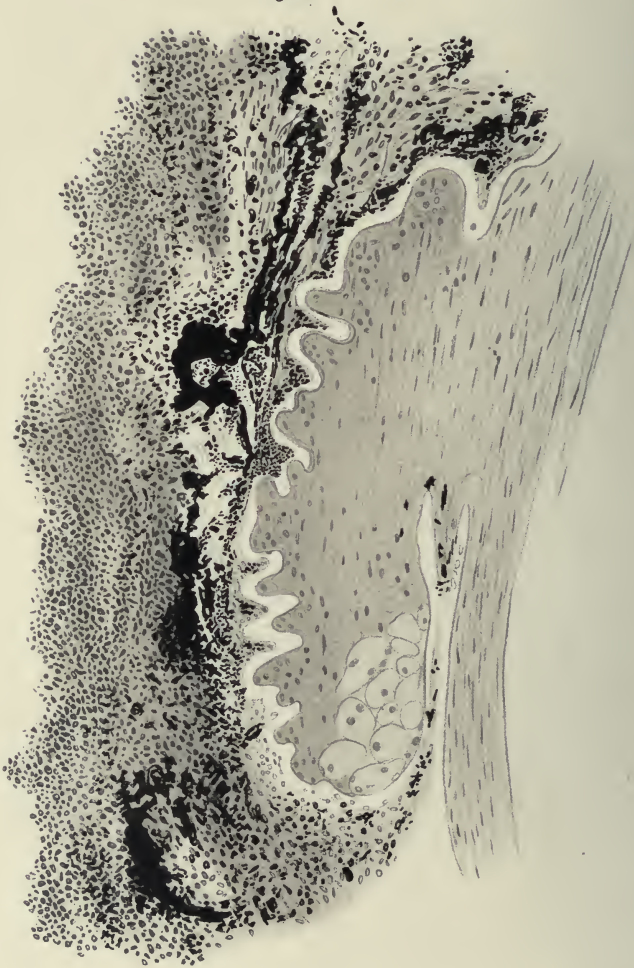


Fig. 3.



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sisting of hypertrophic and hyperplastic unstriped muscle-fibres and spindle-formed connective-tissue cells, originating in the anterior part of the ciliary body, and the diagnosis myosarcoma seems justifiable." An instructive drawing is given of the longitudinally and transversely cut bundles of spindle-cells with rod-shaped nuclei composing the tumor, but it does not convince us that muscle-fibres are a constituent of the tumor.

There is a natural tendency for every one who finds a tumor of the ciliary body with spindle-cells and rod-shaped nuclei to believe that he has to do with a myomatous proliferation of the ciliary muscle. This was true in my case, and my view of the nature of the tumor oscillated between myomatous, sarcomatous, and myosarcomatous, until I had made a more careful study of spindle-celled sarcoma in general and uterine tumors in particular, and had found in the former complete analogues of the tumor structure in my case, which I must, therefore, consider a fascicular spindle-celled sarcoma.

My experience, together with the mostly insufficient histological description of the other cases, with the lack of drawings, and the fact that often different observers differ in their opinion of the same tumor, has led me to the conclusion that the designation of the six tumors as partially or altogether myomatous has arisen more from the subjective conception of the authors, and is not objectively correct.

According to my conviction, the reported cases of spindle-celled tumors of the ciliary body which conform to a definite clinical and anatomical type are all to be considered spindle-celled sarcomata; and the fact that they have been in some cases considered myomata or myosarcomata is due chiefly to the great similarity of the individual spindle-cells and their grouping, to unstriped muscular tissue, and to the fact that they have their origin in a tissue rich in unstriped muscle.

EXPLANATION OF THE FIGURES.

Fig. 1.—A quadrant of the ball with the mottled tumor, the episcleral nodule, the adhesion of the lens-capsule, the partial

absorption of the cortex of the lens, and the break in the pars-ciliaris retinæ. Magnified six times.

Fig. 2.—A portion of the middle of the tumor, with alternating longitudinal and transverse sections of the spindle-cells, the latter grouped into bundles by secondary sheaths.

Fig. 3.—The point of adhesion between the detached and wrinkled lens-capsule and the fibrous tissue and swollen cells which are products of the altered lenticular epithelium.

THE ANTERIOR OUTFLOW CHANNELS OF THE EYE AND THE ARTIFICIAL PRODUCTION OF GLAUCOMA.

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(*With Plate IX. of Vol. XXVIII., Germ. Ed.*)

THE controversy with regard to the passages by which the fluid of the tissues makes its exit from the interior of the eye has continued up to the present time. The only statement that is generally accepted is that the aqueous has its outlet "in front," and that a portion of the liquid of the vitreous gets out of the eye through the lymph-channel of the optic nerve.

In coming to a decision with respect to the experiments bearing upon this point, we must differentiate two things actually distinct, viz.: (1) the *path taken by the body of liquid as a whole*, this passing according to natural laws from the regions of higher pressure (the capillaries) through the tissues and their interspaces into the preformed passages of outflow (the lymph-vessels); and (2) the *channels along which dissolved substances* (including gases) *undergo diffusion.* Such dissolved substances would spread to all parts from which they were absent before or in which they were present before in a less concentrated state.

In experiments upon the path taken by the body of liquid as a whole, we must, for reasons that can be readily appreciated, use substances which are not normally, nor, in fact, under any conditions, present in the eye. In a perfectly dead eye such substances would diffuse uniformly in all directions according to purely physical laws, being affected only by the different degrees of facility for imbibition and diffusion afforded by the tissues. In the living eye this state of things is modified by the natural "tissue-current," and the same is true, though to a less degree, for the recently excised eye, provided that the tissues have not become entirely dead, and all differences in pressure have not been equalized.

Many years ago experiments that I made (Virchow's *Archiv*, vol. lxv., p. 401, and *Arch. f. Augen. und Ohr.*, vol. vii.) proved that potassium ferrocyanide introduced into the cavity of the vitreous passed very readily into the anterior chamber and then left the eye by the same channels as do substances that are introduced directly into the anterior chamber. For the study of these latter channels of exit, in fact, injections into the vitreous cavity are more suitable than injections into the anterior chamber, since in the former case the walls of the latter remain perfectly intact. Hence when I undertook to make a series of experiments intended to demonstrate the effect that substances which, though aseptic, are yet provocative of inflammation, exert upon the anterior channels of outflow in the eye, I determined to introduce these substances into the vitreous. Since in this case the substances employed were such as do not normally circulate in the currents by which the tissue-metamorphosis of the eye is effected, precisely the same conditions were present here as with the injections of potassium ferrocyanide. The latter, in fact, give us the place where we are to look for the effects that substances provocative of inflammation will excite, when introduced into the vitreous.

The experiments were made upon dogs because their eyes, as far as the parts here under consideration are concerned, correspond more closely to human eyes than do those of rabbits, which are generally used. Before describing these experiments, however, I consider it absolutely necessary to describe the channels by which the ferrocyanide introduced into the dog's vitreous leaves the eye. To effect this from one to three drops of a solution of potassium ferrocyanide were introduced as nearly as might be in the centre of the vitreous and, after a varying interval of time, the animals were killed. The eyes were at once enucleated, opened, cut in two, and exposed to the action of ferric chloride dissolved in dilute alcohol. By the blue coloration which is immediately developed, the mode of distribution may then be recognized.

It is evident, in the first place, that this solution undergoes practically no diffusion backwards, but does diffuse into the anterior portions of the vitreous. Spreading from this locality it colors the contents of Petit's canal and of the anterior chamber. It is only later, after six or eight hours' action, that some of the coloration penetrates the lens, beginning at the posterior pole and extending into the posterior cortex.

Generally the ciliary body is also found to be more or less strongly tinged with blue, and so is the adjoining peripheral portion of the iris—the portion constituting Ulrich's peripheral channel of transmission through the iris—which I (*l. c.*) discovered and described much earlier than he did, although I gave a different interpretation to it. In this case the ferrocyanide is found also in the anterior chamber and in such abundance that it seems to me far more probable that the coloring matter had penetrated from Fontana's space than through the iris.

The first stage in the passage of the coloring matter through the wall of the eyeball is manifest after two or three hours under the form of the well-known and much-discussed blue streak in the corneo-scleral junction (Fig. 1). This streak as it makes its way backwards lies farther and farther out in the sclera, and ultimately, at some point behind the equator of the eye, empties into the lymph-space of Tenon's capsule.

This condition of things, which is represented in Fig. 1, is the easiest to obtain, and most observers who have repeated my experiments, seem to have got this stage and nothing more. Much more instructive, however, are the conditions obtained after a somewhat longer time, the most characteristic of which are represented in Figs. 2, 3, and 4.

For, while the streak in the corneo-scleral junction and in the sclera remains as it was, the hindmost layers of the cornea also become colored (Fig. 2), and later on the entire cornea takes on a blue tinge, and the color spreads not only backwards into the sclera, but also into the subconjunctival connective-tissue, in which the main lymphatic vessels run in the direction of the four recti muscles to the angles of the eye and to the retrotarsal folds. We then get pictures like those which Fig. 3 represents.

Later still we find that only the anterior layers of the cornea are colored, the coloration extending from them into the subconjunctival tissue (Fig. 4). The blue streak in the sclera, though still present, does not now extend as far forward as the anterior chamber.

After from eight to ten hours no more ferrocyanide can be found in the eye.

Leber and others, including recently Gifford (these ARCHIVES, vol. xxi., No. 2) regard the results obtained by my method as of little value because of the precipitates produced at the spots where the two reagents first come into contact. But owing to the way in which the eye is cut into and bisected, the reaction is made almost instantaneous. Moreover, I do not know of any other way of fixing the spot to which a substance contained in the eye has penetrated.

From an unprejudiced consideration of Fig. 3, and especially of Fig. 4, there can be no doubt that during life the ferrocyanide had entered the cornea from the anterior chamber. And, as I showed in my earliest papers, the endothelium of Descemet's membrane is not an absolute bar to the passage of dissolved substances from the anterior chamber into the cornea, but is only a means of regulating the passage, since it prevents more from entering than can under normal conditions flow off again at the corneo-scleral junction.

The experiments whose results have been thus shortly summarized were made and published as much as fifteen years ago.

Following are the positive facts resulting from my experiments with respect to the conditions of outflow in the anterior segment of the eye:

1. The aqueous is secreted not only by the ciliary processes, but, to a slighter extent, also is derived from the posterior segment of the eye. Substances which are not normally present in the latter, *i. e.*, pathological substances, are especially apt to get into the aqueous.

2. The outflow from the anterior chamber takes place not only (although it does mainly) from Fontana's space, but a portion of the aqueous, even when the endothelium of Descemet's membrane is intact and the intra-ocular pressure is not heightened, penetrates into the cornea itself and flows in the direction of the subconjunctival tissue. This is particularly the case with substances which are not normally present in the aqueous (and in the anterior portion of the vitreous).

3. Dissolved substances present in the anterior portions of the vitreous also get into the lens, and first into its posterior cortex.

In all these cases it is not a question of preformed closed channels of exit but of a process which goes on in connective-tissue spaces and in the matrix of the tissues. The fluid of the eyes does not get into closed spaces which are lined with endothelium until it reaches the cavity of Tenon's capsule and the efferent lymph-vessels in the subconjunctival tissue.

What has been said holds good only for *dissolved* substances. To solid and formed matters the lens-capsule and Descemet's membrane are perfectly impervious; such matters must either pass through these membranes in a state of solution, or a breach of continuity in the membranes must have taken place. The same is true for cellular elements.

In experiments that I have made in recent years by introducing *into the vitreous aseptic substances provocative of inflammation*, I did not so much concern myself with the changes that these substances excite sooner or later in the posterior segment of the eye; these have been sufficiently described by Leber. My main purpose was to investigate their action in the *anterior* segment of the eye.

Since the matters injected were not traceable directly, and since the changes that resulted from their action required time to make them visible, the latter could be expected to take place only after the lapse of some considerable time. Moreover, I did not intend to produce a transient action which could hardly be demonstrable anatomically, but wished to introduce a difficultly soluble agent which would exert an effect for a longer time. I thought to imitate the conditions exhibited by a malignant growth developing in the vitreous and secreting irritating substances which give rise to reactive inflammation of the tissues; and *hoped at the same time to produce an artificial glaucoma.* I succeeded only to a certain extent, because the reagents used did not secure the permanent effect that I had hoped for and because they acted rather differently than I had expected. Nevertheless, the results are interesting enough, although their main value lies in their clinical and not in their pathologico-anatomical aspect.

The experiments were all made upon dogs under morphine narcosis; and it may be remarked in passing that in this animal the pupil is ordinarily not contracted under these conditions. The eyes were hardened in Müller's fluid and alcohol and imbedded in paraffin, and the sections were stained with hæmatoxylin.

In experiments I. and II., oil of turpentine was injected into the vitreous. This produced detachment of the retina and posterior polar cataract with accumulation of liquid beneath the posterior capsule of the lens; the formation in the anterior chamber of gelatinous exudate which was soon absorbed; and the development of an amorphous exudate in the vitreous. The second case was complicated by an iritis due to injury of the eye. Apart from the consequences due to this accident the anterior part of the eyeball seemed to be but slightly affected by the injections, and the latter *per se* occasioned no glaucomatous symptoms.

The failure here being ascribed to the undue violence of the reaction, a third experiment was made with an injection of a mixture of 1 part of oil of turpentine and 19 parts of olive oil. In this case the injection produced localized opacity and liquefaction of the vitreous with subsequent shrinking of the latter and detachment of it from the retina; and, in the lens, rounded opacities in the cortex and distension of the posterior pole by a coagulated liquid containing globular bodies. *Four slight and transient attacks of glaucoma*, two developing spontaneously and two as the result of the instillation of atropine, and exhibiting the characters of the so-called prodromal stage of the acute inflammatory variety of the disease, occurred in the first two months. No permanent changes were produced, however, and in the third month even the instillation of atropine failed to produce an increase of tension; and after four months, when the animal was killed, the structures in the anterior part of the eye were found to be perfectly normal.

In experiments IV. to VII. a mixture of oil of turpentine, 1 part, and olive oil, 2 parts, was used.

In experiment IV. a glaucomatous attack with T + 2 and the formation of a gelatinous exudate in the anterior chamber set in seven hours after the injection. The anatomical conditions found after killing the animal on the following day were marked œdema of the cornea and distension of Fontana's space and of the adjoining parts of the iris and ciliary body with coagulated fibrinous exudate and red blood corpuscles.

In experiment V. the animal was allowed to live for 50 hours. There were attacks of glaucoma, one 10, the other 34 hours after the injection. The latter produced detachment of the hyaloid membrane, and a grayish non-cellular membrane was formed in the vitreous about the injected liquid. Fontana's space showed no pathological alteration, and, except for marked œdema of the cornea, the anterior portion of the eye was normal.

In experiment VI. one pretty severe attack of glaucoma set in 8 hours after the injection. This lasted 2 days, and was not repeated even when atropine was instilled. The animal was killed at the end of 10 days. The main lesions found were cellular infiltration in the network of the ligamentum pectinatum and apparently also of the optic disc, and beginning detachment and shrinking of the vitreous.

In experiment VII. a transitory attack of glaucoma set in 8 hours after the injection. This was followed by a diminution of tension (T – 1). When the animal was killed 11 days afterwards, the cornea was found to be œdematous and had isolated deposits on its posterior surface; there was an infiltration of round cells in the ciliary processes and in, or rather back of, Fontana's space, and there was hemorrhage in the suprachoroid of the ciliary body; and there were thickening and infiltration of the conjunctiva and some infiltration of the retina and optic disc.

In experiment VIII. pure olive oil was injected. A glaucomatous attack followed 18 hours later and lasted till the following day. An exudate also formed in the anterior chamber; but this shrank and was completely absorbed within three days after the injection was made. These phenomena may both be fairly attributed to the effect of the injected material as such; but the subsequent symptoms, which were those of a violent panophthalmitis, must be regarded as the result of septic infection, which was evidently due to the fact that the oil could not be properly disinfected by heating before it was injected.

The results of the experiments just detailed had been unsatisfactory, because in none was a *permanent* aseptic irritation set up in the anterior portion of the eye, such as would produce a continuous glaucomatous condition instead of producing, as in these cases, merely transient glaucomatous attacks, which were of such a character as to simulate those occurring in the prodromal stage of the disease. With no hope of securing a more permanent and slower action, injections were made with a mixture of mercurial ointment (unguntum hydrargyri cinereum 3.0) with olive oil (7.0) and oil of turpentine (6 drops); the idea being that the finely divided mercury in this "oleum cinereum" would pass slowly into the circulatory channels of the eye and produce a continuing condition of obstruction and inflammation. This "oleum cinereum" was applied in cases IX. to XIII.

In experiment IX., in order to follow the action of the material to its end, the case was watched for 11 months.

On the third day after the injection there was a violent attack of glaucoma which lasted 2 days. Simultaneously there was formed in the anterior chamber a gelatinous exudate, which by the sixth day had contracted to one half its original size. On this latter date there were the first signs of an irido-choroiditis which, at first associated with diminution of tension, subsequently led to increase of tension, the formation of an annular posterior synechia and retro-iridic exudate, and in 2 months to phthisis bulbi. From this time on the eye remained perfectly free from injection and irritation; and it is to be noted that during the whole course of the disease, the irritative symptoms remained very moderate, and that no tendency to the development of a sympathetic inflammation in the other eye manifested itself.

In experiment X. the attempt was made to find what were the consequences of the injection in its earliest stages; hence the eye was enucleated 24 hours after the injection had been made. The tissues were mainly normal except for the presence of a coagulum in the anterior chamber[1] and of scattered round cells in the peripheral layers of the vitreous, in Fontana's space, and in the ciliary processes.

In experiment XI. the animal was killed 48 hours after the injection. The aqueous had already become turbid, although the opacities had not yet had time to gather into a coherent mass. The eye, after hardening, showed coagulation of the aqueous and of the vitreous. Round cells were present in the extreme periphery of the latter (in the portion adjoining the retina), and also, and much more abundantly, in the portions next to the ciliary body, and were also found in considerable number in the ciliary body itself, in Petit's canal, and in the vicinity of Schlemm's canal. There was likewise some cellular infiltration of the retina, and the retina at one spot was detached from the choroid by a non-cellular exudate. The anterior chamber contained a cellular exudate only in its peripheral portions.

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[1] Produced by the material used for hardening the specimen; but its presence indicative, according to Knies, of a change in the constitution of the aqueous.—TRANSLATOR.

The cornea was normal except for the infiltration about Schlemm's canal.

In experiment XII., also, the animal was killed after 48 hours. A lenticular exudate had already formed in the anterior chamber. The vitreous here too had been coagulated by the hardening agent. The contents of the anterior chamber were likewise coagulated, and except in the portion adjoining Descemet's membrane were almost entirely free from cells. The cornea was normal except for a cellular infiltration beginning at its margin and extending from there into the innermost layers of the cornea and into the subconjunctival tissue. Cellular infiltration was also found in Tenon's capsule, Fontana's space, the ciliary body (which was hyperæmic), and the ciliary processes, and from the latter was continued into the vitreous and into Petit's canal. The amount of infiltration diminished from before, backwards. The retina was not detached, but near the papilla was folded, and in this region an amorphous exudate lay between it and the choroid.

In experiment XIII. the animal was killed after 72 hours. A pair of lenticular exudates had formed in the anterior chamber. There were no glaucomatous symptoms. The condition on autopsy was essentially that found in the preceding case. The chief additional feature was an abundant cellular infiltration, which was seated in the connective tissue surrounding the central vessels of the optic nerve and which extended into the vitreous.

Experiment XIV., which consisted simply in the examination of a normal dog's eye, was made to determine how far the appearances in the foregoing experiments were applicable to the eye of man. The only essential difference between the two, which could have any bearing upon the results already detailed, was that, upon the optic papilla of the dog and surrounding the central vessels, there is a flattened cone of connective tissue, containing numerous cells and attached by its apex to the hyaloid membrane of the vitreous. This finding makes it doubtful whether the cellular infiltration found in some of the cases in the optic disc was altogether abnormal or not. In other respects the anatomical conditions are the same in the canine and the human eye, and the two therefore, when placed under the same experimental conditions, should react alike and exhibit the same appearances.

It was my object in making these experiments to demonstrate the passages of outflow from the vitreous and aqueous by means of the changes, which substances capable of provoking irritation and inflammation excite in their passage through the walls of the eyeball. The action must vary according as it is vascular or non-vascular tissues that are affected. Direct tissue-changes might perhaps be expected to take place; furthermore coagulation-processes in the channels of outflow and consequent œdema of the tissues back of them; and—most important and most obvious change of all—local inflammation manifested by cellular infiltration of the parts affected.

From the results of the injection-experiments with the potassium ferrocyanide changes were to be expected in the vitreous itself, especially in its anterior segment, in Petit's canal, in the anterior chamber, in the cornea, in the angle of the iris and its vicinity, and later also in the lens. From clinical experience it is likely that the optic papilla also and its vicinity would be involved, it being understood that our investigation deals with only those changes that are the direct consequences of the injections, and not with those that occur weeks and months afterwards. It should also be stated that obvious pathological changes due to the injection are not to be looked for as soon as those that prove the mere presence of an injected material like potassium ferrocyanide; the former requiring a certain time for their development. The results of my experiments are in complete accordance with this fact.

The mildest of the reagents was olive oil, disinfected with oil of turpentine (1:19). After the primary effect, due to its introduction, had ceased, the eye became perfectly quiet again. The more violent action exerted by the unmixed olive oil is explained by the fact that it was not aseptic, and that the germs and their decomposition products which it contained were not destroyed by admixture with turpentine, as in the other cases. Furthermore, the oil itself, unless perfectly fresh, probably exerts an irritant action, even when it is aseptic, as the products of its *chemical* decomposition (glycerin and fatty acids) are more or less soluble, and decidedly irritant. However, as far as the results of the experiments are concerned, it is a matter of indifference what substance is used for the injections. When a very active material is used, its remote effects come to an end sooner, because the material itself is speedily encapsulated by a membrane produced by coagulation of the tissues in its vicinity.

The first result, clinically speaking, of the injection of dilute oil of turpentine, was, in every case in which the observation could be made at the proper time, an *attack of glaucoma*, setting in after a period of about six hours (rarely more), during which the eye had remained apparently normal. This attack had the character of those occurring in the so-called prodromal stage of glaucoma, and, being always quite transitory in character, escaped observation in cases I. to III., because it had come on and had disappeared during the night. The attack was characterized by the usual signs: marked increase of tension, mydriasis (in spite of the simultaneous use of morphine), and sometimes slight vapor-like opacity of the centre of the cornea.

The fact that the attack did not develop immediately after the injection, is of importance. From the amount of time that it takes for substances to diffuse through the eye (as shown by the previous experiments with potassium ferrocyanide already mentioned), it is evident *that the substances which had been introduced into the vitreous must have already been for some time in the region of the anterior chamber and corneo-scleral junction, and have been exerting their action there, before the attack of which that action was the cause, had taken place.*

The pathologico-anatomical condition found at this time was perfectly negative, apart from the œdema of the cornea and apart from the altered chemical composition of the aqueous and vitreous that was evident from the presence of the small clot in the anterior chamber and the clotting of the vitreous. Nevertheless, the subsequent results of the experiments afford a clear explanation of the cause of the attack.

The first *visible* effects of the injections were processes of coagulation. Since the vitreous and aqueous under normal conditions do not coagulate even under the influence of the ordinary hardening agents, they must be mingled with an albuminous transudate, which under certain conditions may even undergo coagulation during life. But while the albuminous transudate derived from the vessels of the retina, the ciliary processes, etc., makes its way into the vitreous. Petit's canal, and the posterior and anterior chambers, the albuminous transudate derived from the vessels of the corneoscleral junction passes into those interspaces of the tissue that serve for the channels of outflow from the eye. *If the transudate coagulates spontaneously, the channels of outflow are blocked up, and an attack of glaucoma is produced.* After some time the same process that is observed on a large scale in the aqueous takes place on a small scale in the interspaces of the tissues, *i. e.*, the coagulum contracts, and undergoes resorption. In this way room once more is made for the outflow, and the attack of glaucoma ceases. Indeed, it is probable that by the processes of exudation and coagulation the tissue-spaces are actually dilated and the outflow thus rendered freer than normal, since ordinarily after the glaucomatous attack a noticeable reduction of the tension below the normal takes place.

Now, by the blocking up of the anterior channels of outflow the passage of the injected irritants through the latter is cut off, and the action of these irritants temporarily ceases; but after the contraction of the coagulum, if there is any injected matter still present, the original processes may be repeated at the corneo-scleral junction and in the anterior chamber, so that in this way we may have the repeated formation of a lenticular exudate and repeated attacks of glaucoma.

The irritant action is manifested very soon also in the *immediate neighborhood of the injected matter*, and this latter, therefore, becomes encapsuled in a coagulation membrane, and thus its further activity is prevented altogether or very much weakened. The exudate in the vitreous that forms this coagulum springs, to all appearances, primarily from the vessels of the retina. In this regard, there is an apparent contradiction between the results obtained by injecting the non-irritant potassium ferrocyanide and those got by injecting irritant substances, since in the former case a direct exit through the posterior channels of outflow (papilla and optic nerve) is generally impossible to demonstrate. But this is explained very readily by the fact that non-irritant solutions *simply pass through* the anterior channels of outflow, while the irritant solutions produce there an obstruction to their own passage, so that their path is deflected into other channels which, under ordinary circumstances, are but little, if at all, employed. In the latter case, therefore, the corneal margin and the optic-nerve entrance would act vicariously for each other.

The *subsequent course* depends largely upon the intensity of the irritation. If the latter is slight (*e. g.*, in case III.), there need not necessarily be any clinically-visible, spontaneously-coagulating, lenticular exudate formed in the anterior chamber. If the irritation is more violent, an exudate of this character develops very speedily, in some cases so speedily that no preliminary increase in tension can be made out (experiments with the "oleum cinereum"). Furthermore, in the course of about two days, if oleum cinereum is used, but later if the injections are made with dilute oil of turpentine, a cellular infiltration is produced in the vicinity of Schlemm's canal (and to a less extent in the retina and optic papilla), which causes disintegration of the tissues, and ultimately, if the irritation keeps on, develops into chronic iridocyclitis and iridochoroiditis. The fact that these last-named processes took their origin from the angle of the anterior chamber was very evident, from several experiments.

If, on the other hand, the irritation is sufficiently slight, it may happen, after the prodromal glaucoma has run its course, that everything becomes, apparently, normal once more, and remains so for a longer or shorter space of time, until some fortuitous exciting cause produces new symptoms. In this connection, experiment III. is of extreme interest, since in the course of it two spontaneous attacks of glaucoma and two that were produced by atropine were observed. In my opinion, the second spontaneous attack was caused by spontaneous rupture of the membrane that encapsulated the drops—a rupture which could be demonstrated ophthalmoscopically, and as a result of which the remainder of the encapsulated irritant liquid was enabled to exert a renewed action. *After this had taken place, no subsequent attack could be produced, even by atropine.* The ultimate anatomical state was normal, especially in the region of the anterior channels of outflow, the only exceptions being the vitreous, which, being subjected to the direct action of the injections, had become completely shrunken, and the posterior cortex of the lens, which, being close to the point where the drops were injected, showed cataractous changes (Fig. 7).

There is quite a marked difference in the behavior of the eye after the injection of dilute oil of turpentine (cases IV.–VII.) and oleum cinereum (X.–XIII.). The former speedily produced a considerable elevation of tension. On the other hand, the spontaneous coagulation of the aqueous was but slightly pronounced. With the oleum cinereum, on the contrary, an abundant coagulating exudate formed in the anterior chamber, and cellular infiltration could be made out as early as the beginning of the second day. The action of the dilute oil of turpentine was at the outset limited almost entirely to the anterior channels of outflow, and the experiments with it, therefore, gave very well indeed the clinical picture of a prodromal attack of glaucoma. The action of the oleum cinereum was quicker, more widespread, and more violent, and hence better suited for demonstrating the anterior and posterior channels of outflow by the cellular infiltration it caused.

The anatomical conditions found after the action for a short time of stronger irritants (turpentine and olive oil 1:2) —namely, cellular infiltration of the vicinity of the angle of the anterior chamber and the optic papilla,—and also those found after injecting oleum cinereum, correspond completely to the conditions found in *glaucoma imminens*, which I described in my first publication upon glaucoma and of which Nettleship afterwards published several cases (sarcoma of the choroid before the beginning of the glaucomatous symptoms). In connection with this anatomical state there is no increase of tension; in fact, according to Nettleship, there is actually a decrease of tension.

Nevertheless, in my experiments the subsequent course was not that of a glaucoma, since the action of the liquids used by me was obviously different in a qualitative way from that of the products of tissue-metamorphosis that are the efficient agents in human glaucoma. These latter products, which we may conjecture to exist in certain cases (sarcoma and glioma, a cataractous lens undergoing solution in the vitreous, certain detachments of the retina, etc.), obviously act less violently but in a permanent fashion, and thus *inter alia* produce in the angle of the anterior chamber an adhesive form of inflammation of a different kind from that caused by oil-of-turpentine injections or by the products of metamorphosis of the bacteria of suppuration. Perhaps repeated injections of very dilute oil of turpentine might have accomplished the same result.

For those who are not so prejudiced as to cling obstinately to preconceived ideas, the pathology of glaucoma will be clear from the results of these experiments and of the anatomical conditions found in the later stages. If we exclude glaucoma simplex, which runs its course without elevation of tension and without any inflammatory symptoms, and which is only an optic nerve atrophy with a large physiological excavation, a disease in fact that v. Graefe originally distinguished from real glaucoma under the name of "amaurosis with excavation of the optic nerve"—if, I say, we exclude glaucoma simplex, *glaucoma is an irido-cyclitis in which the anterior channels of outflow of the eye are occluded, temporarily at first, afterwards permanently;* the permanent occlusion being due to adhesion of the periphery of the iris to the cornea, *a condition which in the advanced stages is never absent.* Hence it is too that the symptom of increase of intraocular tension plays such an essential part in all the varieties of true glaucoma, although it is by no means continually present, and may actually alternate with diminution of tension.

So long as the inflammatory irritation in the anterior outlets of the eye is a transient one, so that it is still possible for everything to return to its normal condition, only prodromal attacks occur, and the angle of the anterior chamber remains normal; and it is not until an anatomically demonstrable permanent obstacle to outflow is produced by the development of a peripheral anterior synechia that the real glaucomatous attacks set in. Then nothing but the making of a new channel of outflow from the anterior chamber into the subconjunctival tissue, which is effected by means of the peripheral section at the corneo-scleral junction, is able to produce a return of equilibrium in the conditions of tension in the eye. It does this in a great number of cases. Should, however, an obstacle to the outflow still exist—and this may possibly be due to the fact that the processes of coagulation in the channels of outflow still keep up,—we then get the *malignant* forms of glaucoma which generally run a very acute course.

Glaucomatous irido-cyclitis is distinguished from the ordinary irido-cyclitides by the fact that in the latter the nocuous element has from the outset its seat in the iris and ciliary processes, while in the former it circulates in the vitreous and aqueous and in its exit from the eye causes an inflammation of the iris and ciliary body starting from the region of the angle of the anterior chamber. It must be admitted that in the genuine glaucomas we can make no definite statement in regard to the nature of this element; while in the secondary glaucomas, *i.e.*, in those for which we know an exciting cause, whether it be a malignant tumor, a luxated lens, or retinal hemorrhages, we must assume the existence of such an element circulating in the vitreous.

The *injurious action of mydriatics* is explained by the fact that they cause hyperæmia of the fundus, and consequent acceleration of tissue-metamorphosis there and in the vitreous, so that the nocuous elements contained in the latter are hurried along faster and in greater quantity towards the channels of outflow of the eye. When the nocuous element no longer is present (*e.g.*, in experiment III. after all the oil of turpentine had been discharged), instillation of atropine fails any longer to produce a glaucomatous attack.

A few words in regard to the other anatomical conditions found in my experiments.

The *aqueous*, even in cases in which it did not undergo spontaneous coagulation, showed the formation of a coagulum in the hardened eye, a proof that it was pathologically altered in composition, and particularly that it contained albumin. The coagulum when formed spontaneously was in the early steps nearly devoid of cells; in the later stages it contained them in greater or less amount. The round cells obviously come in but very small part from the anterior surface of the iris. The larger portion lay mainly upon the anterior surface of the exudate directly in contact with Descemet's membrane; the greater amount being along the lower half of the cornea, and the cells being especially abundant in Fontana's space, giving the appearance as if they had been filtered off from the latter. They obviously originated mainly from the region of the posterior chamber.

Apart from one case (IV.) in which a similar cellular fibrinous coagulum was contained in the tissues of the rest of the iris, one (VII.) in which the anterior endothelium of the iris was partially detached, and one (XI.) in which there was œdema of the posterior epithelium, the *iris* was practically normal. Changes in the iris, therefore, cannot be the determining cause of the fibrinous character of the aqueous.

Where there has been increase of tension and for some time afterwards, thickening of the *cornea* is regularly found. This thickening is often obviously caused by œdema and for the most part is distributed very uniformly over the whole surface of the cornea. Only in a few cases could a delicate, vapor-like opacity of the centre of the cornea be made out by clinical observation. Since in the experiments the cornea remained absolutely intact, no assumption is possible except that after the occlusion of the channels of outflow at the corneal margin a certain quantity of liquid still made its way by diffusion from the anterior chamber into the cornea, and when the obstruction was removed came out once more either from the anterior layers (towards the subconjunctival tissue) or the posterior layers (through the sclera) (*cf.* Figs. 2, 3, and 4). Thus my view, enunciated almost 20 years ago, that the endothelium of the posterior surface of the cornea forms no absolute bar to the passage of liquids, but merely regulates their passage, finds renewed confirmation.

It is not surprising that, after an encapsulated drop of irritating liquid has remained for some time in the vicinity of the posterior pole of the *lens*, lenticular opacities especially in the posterior cortex should occur. It is a matter of interest that in experiment II. punctate cataractous formations, could be observed in the anterior capsule also, it being well-known that anterior capsular cataract is a clinical evidence of a complicated cataract, *i. e.*, of one associated with pathological changes in the fundus.

The changes in the *vitreous*, apart from the coagulation membrane that formed about the injected substances, consisted essentially in an imbibition with a coagulable transudate, so that the entire vitreous became solid under the influence of hardening agents. In the earliest stages the coagulated vitreous was almost devoid of cells; later an infiltration with round cells developed, beginning in the most peripheral layers adjoining the retina and ciliary processes.

The material for the coagulation of the vitreous is in part that which is produced by the retina, and serves for the condensation of the hyaloid membranes. If the irritation is but slight and transient (experiment III.), the vitreous and hyaloid separate from the retina and may contract to very small dimensions without causing further changes in the latter. The place of the vitreous is then occupied by liquid.

If the irritation is more intense, it makes itself felt upon the *retina* even before the shrinking of the vitreous. The innermost layers of the retina are infiltrated with round cells, which originate in the most peripheral layers of the vitreous, and are generally most abundant about and upon the optic papilla. When the irritation lasts a long time, total detachment of the retina occurs (experiments I. and IX.). This, however, does not owe its development simply to the infiltration and subsequent retraction of the inner layers of the retina. Its beginnings are manifested at a very early period, before there can be any idea of such infiltration and retraction taking place, in the vicinity of the optic papilla, where folding of the retina and the presence of a coagulated exudate between these folds and the choroid (cases XI., XII., XIII.) prove the existence of a commencing detachment of the retina.

This concentration of the exudate about and upon the *papilla* compels the conclusion that channels of outflow are present in the interstitial tissues of the optic papilla, and that similar processes take place in the latter as in the tissues of the anterior channels of outflow. The accumulation of liquid about the papilla between the retina and choroid would hence be originally a symptom of engorgement. This is the spot in which the halo is found when there is glaucomatous excavation. In our cases a total detachment of the retina developed out of these circumpapillary changes; in glaucoma an atrophic spot, resulting from an adhesive inflammation, forms in the choroid; hence the so-called halo.

While evidences of inflammation were found in the retina and papilla, these evidences did not extend any great distance behind the lamina cribrosa. The outflow from this point therefore appears to take place not so much in the optic nerve itself as in the tissues surrounding the central vessels and in the spaces ensheathing the nerve.

The *choroid*, in the days immediately succeeding the injection, showed no visible changes.

In conclusion, the results of my researches may be briefly recapitulated as follows:

The experiments had for their object (1) to *render visible the channels of outflow from the eye (especially those of the anterior chamber) by means of the inflammatory changes which aseptic agents provocative of inflammation, that had been introduced into the vitreous, would excite* in the course of their passage through these channels. The results agreed with my former researches with indifferent substances, inasmuch as they both showed that the outflow (which does not occur in closed cavities, but in the interspaces of the tissues), takes place mainly from the angle of the anterior chamber through the sclera to Tenon's capsule, and to a less extent through the unchanged cornea into the subconjunctival tissue. But since, as a result of the changes at the corneo-scleral margin, an occlusion, although but a temporary one, took place in the anterior channels of outflow, a further result occurred in these cases which did not occur when indifferent substances were used, and which consisted in the fact that the existence of the posterior channels of outflow was likewise made evident by means of inflammatory processes taking place in them. Of course, it is understood that this holds good for the passages of outflow only for liquid which has got into the vitreous chamber, and not for that which comes from the tissue of the choroid, for example. The starting-point of these posterior channels of outflow proved to be, as it is generally supposed to be, the optic papilla, and especially its interstitial tissue.

2. It was the object of the experiments to *produce inflammatory processes of an adhesive nature in the anterior channels of outflow*, and produce consequently *artificial glaucoma* by means of the inflammatory agents as they passed out through these channels. This was only partially successful. Only a temporary occlusion of these channels, in other words, only prodromal glaucomatous attacks, could thus be produced. The subsequent course either was that the inflammatory agent gradually disappeared and thus a cure was produced, or that the agent acted differently from the way in which it had been anticipated that it would act; in fact, cellular infiltration, and not a simple adhesive inflammation, set in in the spots affected. A *continuous occlusion* of the anterior channels of outflow, such as is characteristic of fully developed glaucoma, could not be secured.

Nevertheless the results of the experiments are of the greatest importance for the pathogeny of glaucoma. For the first time the attempt was made deliberately and success fully to produce what are clinically speaking prodromal attacks of glaucoma, and what from the point of view of anatomy is an isolated inflammation of those spots, namely, of the vicinity of the *sinus of the anterior chamber and the optic nerve*, which in uncomplicated glaucoma are likewise the only ones that show pathological changes—to produce in short the picture of glaucoma imminens, as I have already described it in man, consisting in cellular infiltration of these two regions occurring before the slightest clinical symptoms were yet present. Nettleship described a similar picture occurring in the case of eyes with sarcoma of the choroid before the development of glaucomatous symptoms. Obviously, in order to ensure the development of a full-fledged glaucoma, the inflammatory agent *must act continuously*, *but less intensely* than did the material that I used, *i.e.*, must probably act like the substance secreted by a growing malignant tumor. Whoever succeeds in finding such a continuously acting inflammatory agent will have it in his power, by introducing this substance into the vitreous, to produce a fully developed glaucoma. This seems, however, scarcely necessary, since every sarcoma of the choroid, every lens displaced into the vitreous by couching is really fully equivalent to an investigation of this sort, and could not possibly be made more free from extraneous influences if it were done experimentally.

It is hoped that with the results here given the theory of the neurotic nature of glaucoma has been done away with once for all. An agreement upon this point could never have been reached as long as the confusion was maintained between things so distinct clinically and anatomically as the so-called glaucoma simplex and the inflammatory forms of glaucoma. A. v. Graefe's clinical acuity first recognized their non-identity, and the results of subsequent anatomical investigations have more and more confirmed his view. If we call *glaucoma simplex* what it really is, *i.e.*, *optic-nerve atrophy with excavation*, and the *true forms of glaucoma* we call *irido-cyclitis anterior* (*i.e.*, a form in which the exudate is found mainly on the anterior aspect of the parts), an inflammation which may occur in every possible degree of acuity

Taf. IX.

Fig. 1.

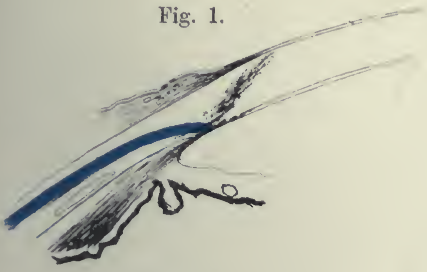


Fig. 2.

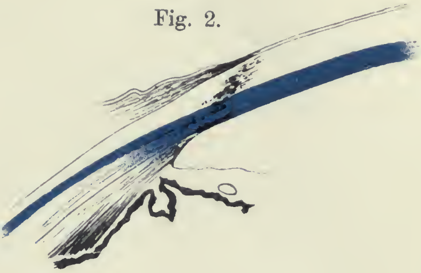


Fig. 3.

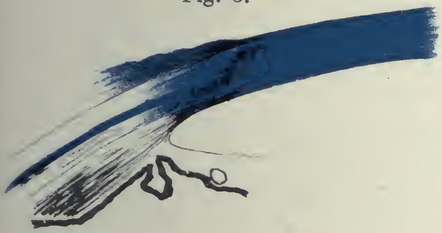


Fig. 4.

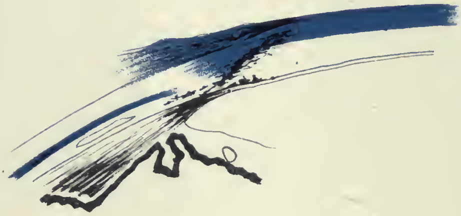


Fig. 5.

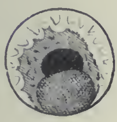


Fig. 6.

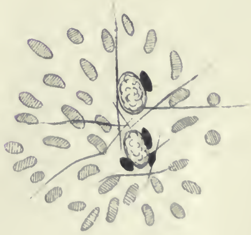
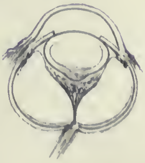


Fig. 7.



VERLAG VON J. F. BERGMANN, WIESBADEN.

and intensity, we then have designations in which the clinical symptoms and the anatomical conditions are strictly correlative; in short, we have well known and readily recognizable clinical pictures representative of diseases that have a real existence.

EXPLANATION OF THE ILLUSTRATIONS.

FIG. 1–4.—Different successive stages of the passage through the anterior channels of outflow in the eye of potassium ferrocyanide that has been introduced into the vitreous.

FIG. 5.—Gelatinous exudate in the anterior chamber in experiment I., 4 days after the injection of oil of turpentine into the vitreous.

FIG. 6.—Beginning of anterior capsular cataract in experiment II., 3½ months after the introduction of the oil of turpentine into the vitreous. In this case on the fourth day a plastic hemorrhagic irido-cyclitis had developed as a result of traumatism.

FIG. 7.—Section of eye No. III., 4½ months after the introduction of oil of turpentine diluted with olive oil.

CONTRIBUTIONS TO THE CONGENITAL ANOMALIES OF THE FUNDUS OCULI.

By Dr. MITVALSKY, of Prague.

Translated (abridged) by Dr. J. A. Spalding, Portland, Me.

(With two chromo-lithographic Plates, X. and XI. of Vol. XXVIII. Germ. Ed.)

I.—TWO CASES OF PARAMACULAR COLOBOMA OF THE CHOROID, WITH TUBULAR FORMATIONS.

HIRSCHBERG has shown (*Berlin. klin. Wochensch.*, 1892, p. 327) that cylindrical formations in the vitreous are often mistaken for encapsulated cysticerci, and even appear as such in literature, amongst which cases he cites those by Liebreich and v. Graefe (*v. Graefe's Archiv*, i., p. 348, and ii., p. 263), to which may be added that by Teale (*R. L. O. H. Reports*, vol. v., p. 310). In the May number of last year's (1894) *Centralblatt*, he returns to the subject, and proves that the first two cases must be connective-tissue formations in the vitreous.

Hirschberg was also the first to dissipate the myth of the spontaneous cure of cysticercus of the interior of the eye, and to place the diagnostic error where it belongs. He communicates in the same paper an additional case of a congenital formation in the vitreous produced by the persistence and condensation of the connective tissue surrounding the hyaloid artery. His chief diagnostic points are: Discoloration of the iris; a bluish cylinder which extends from near the papilla through the vitreous, is fixed behind by tent-like fibres, and terminates in front in minute fibres and membranes which almost reach the posterior capsule of the lens; and, finally, loss of vision in the affected eye, with a dark spot in the field of vision, and strabismus.

I will now describe two similar cases, which vary somewhat from those so far described, and which I have had a chance of watching for many years.

CASE 1.—Mr. A., aged twenty-four, has always had a defective left eye, which deviates outward 40°.

The iris is of the same color as the other.

Media transparent, papilla very white, atrophic, excavated, slightly pigmented, and exhibiting a conus (Fig. 1). The upper temporal quadrant shows a congenital defect, which seems to be a paramacular coloboma of the choroid, and of peculiar form, as shown in the figure. The entire coloboma is devoid of choroidal vessels, but contains retinal vessels, and is well defined from the remaining fundus of the eye, which is normal in color.

The angle between the two portions of the coloboma shows a cylindrical structure, with one end in the retina, and the other, larger, extending into the vitreous. It is about the shape of an almond, is about half the diameter of the papilla, and near the retina gives off an offshoot, but as it extends forwards has no branches. There is no trace of vessels, but slight pigment around the place of attachment. The refraction of the retina at the point of implantation is + 1 D. S., and that of the summit of the growth + 6 D. S.

The retinal vessels show but few changes, and these can be studied in the sketch. The chief rarity is that the inferior retinal artery arises from the upper part of the disc, and bends downward properly after a short excursion.

Vision $\frac{1}{30}$, and field concentrically contracted.

I have followed the case for years, and there has never been any change in the ophthalmoscopic image of the eyes.

CASE 2.—The second case is that of a girl of fifteen, who has a bridged-over coloboma of the iris of the right eye downward, so that she has a double pupil. Ah of + 2 D., and a normal fundus oculi. Vision $\frac{6}{10}$.

The left eye diverges 20°, but the iris is of the same color as that of the other eye. The optic papilla (Fig. 2) in the erect image is oval, and covered with a stripe of whitish-gray tissue, which begins in the porus of the papilla, covers the exit of several of the retinal vessels, and extends toward the periphery of the fundus of the eye, decreasing in diameter at first, and then enlarging as it enters upon a defect in the choroid of a figure-of-8 shape. The choroidal coloboma offers attachment for a greenish-gray cylindrical formation, which then extends into the vitreous. Refraction of papilla + 1 D. S.; that of the coloboma + 3 D. S.; of the body of the neoplastic structure about + 8, and the extreme summit + 10 D. S.

Vision $\frac{6}{20}$, and field defective on nasal side.

I will not attempt to establish any hypothesis concerning this structure, but I think that it is like those described by Hirschberg and Dimmer,[1] because they, as well as v. Graefe and Teale, speak of similar structures in the vitreous. In all of these cases, however, the neoplasm arose from the papilla, so that they must have owed their existence to the hyaloid artery and its sheath. But in both of my patients it arose *at a distance from the papilla*, amidst defects in the choroid, so that it would seem a new sort of congenital defect.

It should also be emphasized, in opposition to Hirschberg, that both irides were of the same color; the outer end did not terminate in an awning-shaped expanse, and there was no central scotoma, only peripheral contraction of the visual field.

II.—A CASE OF CHOROIDAL DEFECT, WITH TENT-LIKE PSEUDO-MEMBRANE IN THE FUNDUS OF THE EYE, TOGETHER WITH MALFORMATION OF THE PAPILLA.

Miss L., aged eleven, has divergent squint of O. D., beginning two years ago, and subsequent loss of sight. The left eye is normal.

The right eye has vitreous opacities, and a curious ophthalmoscopic image (Fig. 3). There is no papilla, but at its situation begins a yellowish nine-pin-like structure lying horizontally, with a blunt, round, small end at the papilla, and then extending out to and across the macula to the temporal periphery of the eye. Whatever remains of the original papilla is embraced by the small end of the neoplasm, because the retinal vessels originate at that spot alone. Attention should be drawn to the difference of level between the horizontal structure and the rest of the retina, the refraction of the former being + 4 D. S., while that of the retina is barely + 1 D. S.

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[1] These ARCHIVES, vol. xiv., p. 72.

We next look at the shiny white tissue lying in front of the horizontal structure, and terminating in a tent-like appearance, which is a rarity in ophthalmoscopic images. It seems to be attached to the middle of the nine-pin-shaped structure, and towards the termination in a tent shape, projects well into the vitreous and toward the equator of the globe. The extreme fibres spread apart, and permit a clear view of the fundus of the eye.

Coursing down from this white patch is a shiny line extending farther along into a slight serous detachment of the retina. Directly above the tent-shaped termination is a defect in the choroid, seemingly connected with the horizontal structure, but proved by the refraction ophthalmoscope to be an entirely different structure—a sort of coloboma of the choroid.

Finally, we observe the ring of brownish pigment surrounding in its embrace the nine-pin-shaped body, the coloboma of the choroïd, and part of the whitish shiny tissue with the tent-like termination.

There can be no doubt, that the nine-pin-shaped figure is formed by the retina, mechanically raised from its choroidal foundation, and that the cause of the whole is the fibrous matter, which in its turn must be regarded as the sequence of pathological processes in the fœtal connective tissue of the secondary ocular vesicle. The fibrous substance originated in the region of the papilla, and in the course of growth of the eye produced traction upon the papilla and the retina. At the age of ten, vitreous fluidity was excited by unknown means, together with a serous retinal detachment in the inferior quadrant of the retina, and this was what first called attention to the pathological condition of the eye.

III.—A TYPICAL CASE OF PERSISTENT HYALOID ARTERY.

Miss K., æt. thirty, has never seen well with O. D., and three years ago suffered from papillitis of O. S., with vision reduced for many weeks to $\frac{1}{4}$, followed by gradual restoration of the normal conditions and of good sight.

O. D. externally normal, and without deviation. Slight calcareous formation in periphery of lens. A whitish strand begins near the rear of the lens, and extends to the papilla, where it spreads out into a globular formation, which cannot be well seen unless the eye is turned up and in, for it runs so directly from the lens to the papilla that its course cannot otherwise be followed.

Examination in the erect image (Fig. 4) shows a well-formed papilla with much pigment on one side, and on its lower half the greenish termination of the hyaloid artery. The rest of the fundus seems normal. Myopic astigmatism — 3 D. C. ⌢⌣ — 2 D. S. The refraction of the globule is about — 1 D. S., and the summit of the cord is best seen with + 11 D. S. Vision with best lens, $\frac{1}{3}$. Vision of O. D. normal with lens.

The case needs no commentary.

Fig. 1.

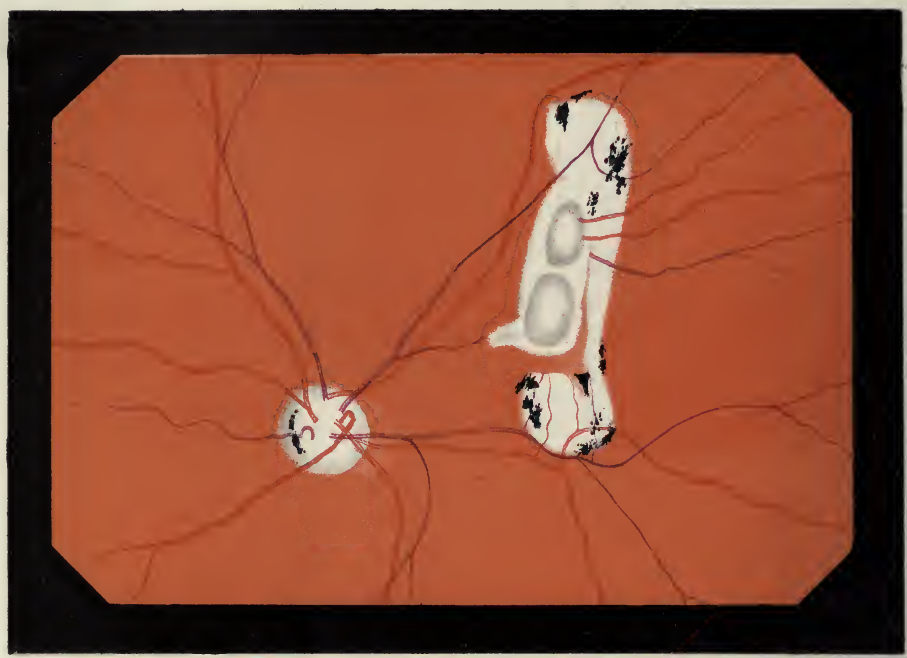

Fig. 2.

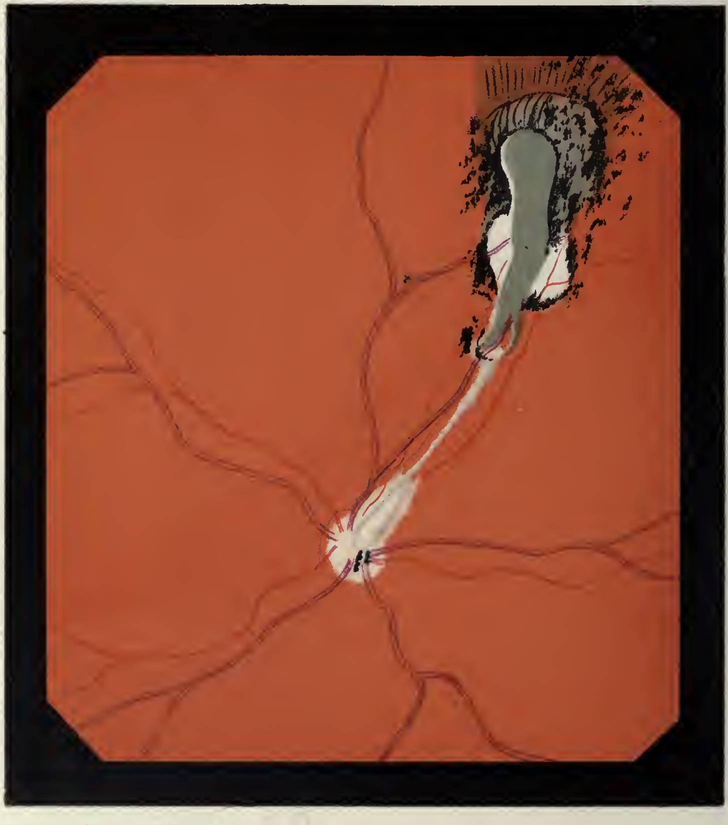

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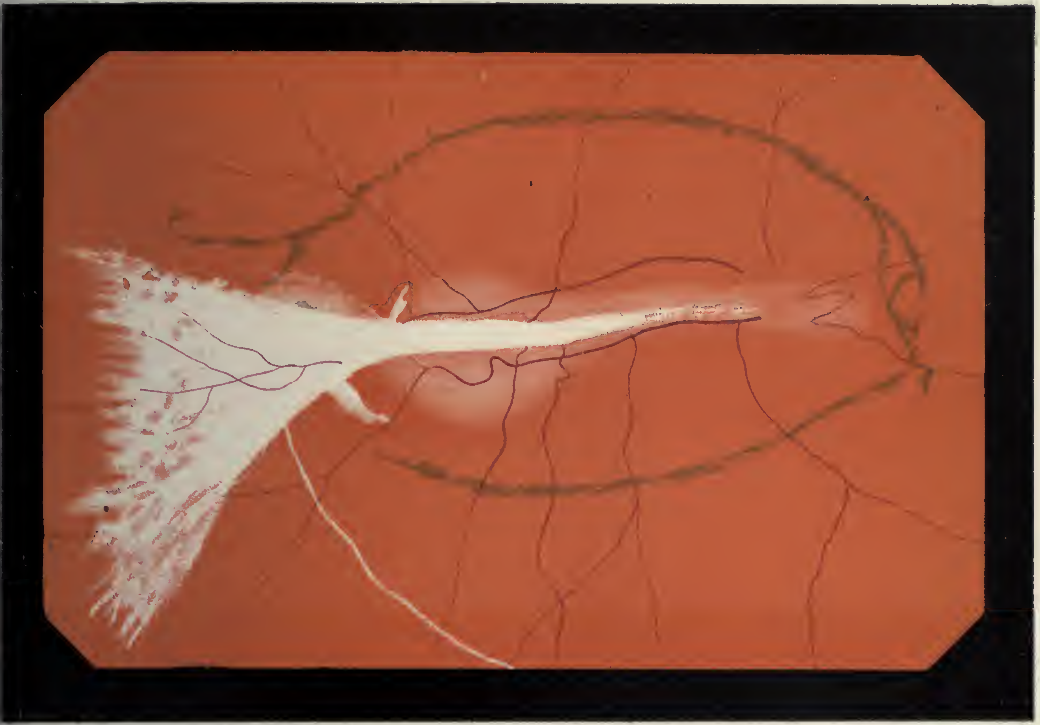
Fig. 3.



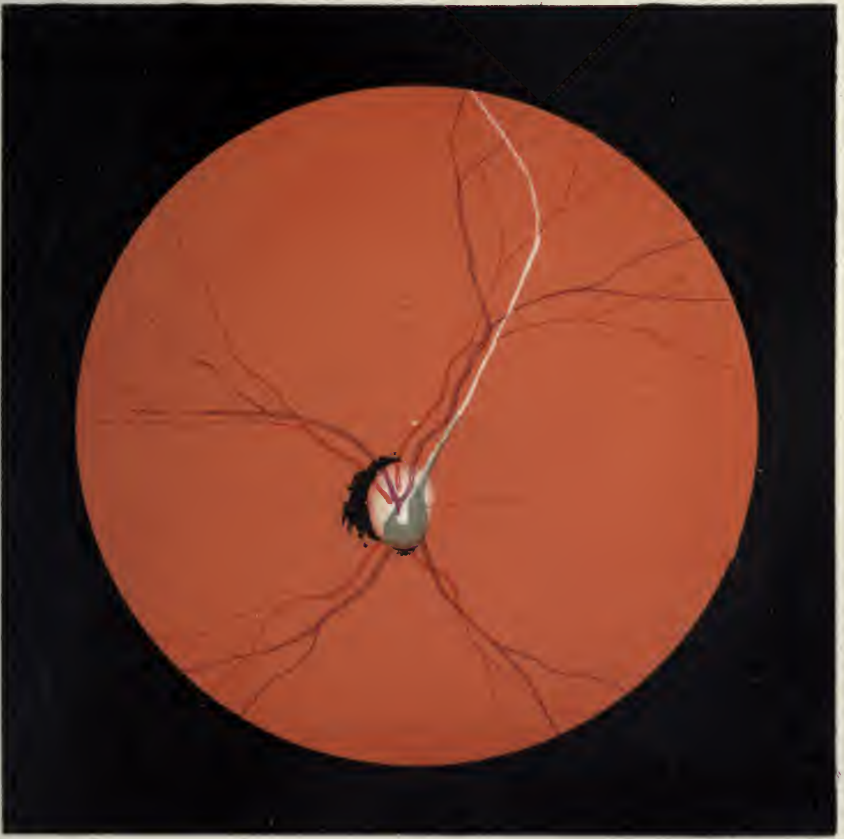


Fig. 4.

VERLAG VON J. F. BERGMANN, WIESBADEN.

A CONTRIBUTION TO THE OCCURRENCE AND ORIGIN OF CONGENITAL ANOMALIES OF THE EYES.

By Dr. HUGO WINTERSTEINER, of Vienna.

Translated (abridged) by Dr. J. A. Spalding, Portland, Me.

(*With one chromo-lithographic Plate XII. of Vol. XXVIII. of Germ. Ed.*)

OUR knowledge of the developmental history of the eye is still incomplete, partly owing to the obstacles raised by the nature of the material to be examined, and partly to the difficulty of deciding what the specimens actually mean. There is, however, a way in which we can enlarge our knowledge of these processes, and that is by observing and analyzing congenital malformations of the eyes, for they offer new keys to the anatomical relations of the fœtal organ, or they confirm the experiences of histological studies, or if the latter cannot be brought into unison with one another, they excite us to new researches. Hence every new case of congenital anomaly which throws any light on our present knowledge, or confirms the same, excites our immediate interest. For all these reasons, I beg leave to report the following case:

Adolph W., æt. thirteen, suffered from inflammation of the eyes when three years old, and was treated for a year. His eyesight has always been poor, and he has not been sent to school. Two years ago he observed that the left eye was the better of the two, and as the right grew worse he came for an opinion, February, 1893.

Congenital epicanthus, no strabismus, both corneæ very hazy, iris and pupils normal. After dilating the right pupil, the following changes were seen :

Simple illumination shows an opaque, glittering mass in the pupillary field, with cords extending above and below (Fig. 1).

The outer cord lying in front of the other gives off a second at right angles, which extends backward to the retina, branching as it passes along. Most of the branches are so tense that, on moving the eye to and fro, no motion in them is perceived. Finally (Fig. 2), there are two horizontally situated, grayish sort of furrows, which extend backward and are inserted into the lower margin of the papilla. The one running toward the nose carries an enlarged vein on its surface, whilst the other has a vessel so small that whether a vein or artery cannot be determined.

The papilla is very white, with an ill-defined border, but is not swollen or excavated. Its vessels are nearly normal. A few patches of pigment are scattered about it. The lower vein passes out a short distance, then turns sharply above the level of the retina, and sinks back again. At one spot it lies close to the neoplastic tissue in the vitreous.

The fundus is normal, only there is no trace of a macula. Near the upper margin of the papilla is a white streak, about the size of a vessel, which passes off into the periphery without much change. It is bordered above in part of its course by a line of pigment. This streak is actually behind the retina.

The upper, inner quadrant of the retina is marked by a similar whitish streak, which is, however, much more irregular in outline.

The globular structures in the vitreous are well depicted in the figure and need no further description. Owing to the haziness of the cornea and the poor control of the eye, the refraction of these masses could not be exactly determined.

Fingers were counted at 2 *m.*, O. D., excentrically. The field could not be accurately tested, but an offhand test showed nearly total loss in the upper half. Vision O. S., $\frac{20}{50}$, and Jaeger 3 at 16 *cm.*, but no improvement by glasses, owing to irregular astigmatism.

Here, then, we have a globular connective-tissue opacity in the vitreous, extending toward the periphery of the globe, retina, and papilla, in the form of connective-tissue bands and cords. The papilla is white, the vessels dislocated, and the retina interspersed with white striæ and pigment.

This condition of affairs may be congenital or due to disturbances of development, or to intraocular hemorrhages or similar conditions.

Inflammation may be excluded, in my opinion, because a part of the alterations—the epicanthus, the abnormal vessels, and the tortuous vein—will not fit into such an explanation. Moreover, the vitreous changes are not such as we are accustomed to regard as inflammatory in origin, for inflammatory products so abundant as to produce such globules and cords are invariably associated with irido-cyclitis, deposits on the capsule, and vitreous opacities, of which there are no traces here. The only possible inflammation may be the so-called retinitis proliferans, in which, as the result of hemorrhage, we see connective-tissue neoplasms in the vitreous and retina. But in all of the cases of this disease which I can recall, the ophthalmoscopic image varied completely from that in the case now under observation.

Hence it seems that the condition here depicted is either a congenital anomaly or defective development, or intra-uterine inflammation, and this view I will try to substantiate by the symptoms.

The vascular abnormities must be congenital or due to defective development of the vessels, because they cannot be explained by subsequent obliteration or thrombosis or secondary enlargement, since their origin from the papilla is abnormal. More than that, the absence of a macula, and the passage of a large vessel through its locality, shows a congenital defect.

Mention must also be made of the vein which, coming from the periphery of the retina, arises just below the macula region above the level of the retina, pushes into the vitreous, and then, bending in hook-shape, returns parallel to its former course in the retina, and then passes to the macular region and papilla. The only cases of vascular formation accessible to me were in *front of the papilla* and *arterial*. The only case resembling mine is one by Hirschberg,[1] in which the retinal veins formed a vascular network on the papilla and partly to one side, but these did not press forward into the vitreous, but lay on a level with the retina.

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[1] *Centralbl. f. Augenhlkde.*, 1883, p. 289. Note to a paper by Czermak on vascular formations in the vitreous.

Czermak's opinion, that these are the remnants of an embryonal vitreous arcade, originating from anastomosis and ramification of the embryonal vitreous vessels, seems to be appropriate also to the appearances in my patient.

The appearances in the vitreous are congenital, though what they mean is doubtful, as I find nothing analogous in the literature at my command. Those most resembling it have been described by Eversbusch[1] and Dimmer,[2] though theirs were smaller, consisting in a tent-like detachment of the retina, extending into a connective-tissue prolongation to the posterior capsule of the lens. Vessels were given off to the retina. Eversbusch thought that the formation reached the capsule, whilst Dimmer was of the opinion that a former junction had been destroyed. Both authors speak of the condition as being a remnant of the hyaloid artery.

Beside being sure that my case is congenital, I also insist that it is not a persistent hyaloid, or a Cloquet's canal. For these never appear simultaneously, and I have never heard of a Cloquet's canal with vessels, to say nothing of veins, on its surface. Ricchiardi has, however, discovered[3] projecting veins in the vitreous, up to the third month of fœtal life, and there was distinct anstomosis between the capsular artery, and the peripheral vessels of the vitreous.

We can therefore imagine that, owing to undue influences, these fœtal vessels did not retrogress as usual, that their walls proliferated, and were eventually enveloped in a connective-tissue mantle, as shown in the figures. The arteries can be explained in the same way as we explain their appearance in neoplasms in the vitreous.

Jaeger was the first to call attention to cords in the retina,[4] but since then only few similar cases have been depicted (chiefly by Perrin, Hirschberg, and Magnus), and all entitled retinitis striata. Other cases so far published refer to similar images, but to dissimilar processes, as is plain to any careful observer.

The nature of these striæ is still in the dark, because no one has yet been able to watch their origin or growth.

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[1] *Mittheilungen aus d. Univ. Augenklinik. z. Muenchen*, 1882.
[2] These ARCHIVES, vol. xiv., p. 73.
[3] Nagel's *Jahresbericht*, 1872, p. 73.
[4] *Atlas*, 1869.

Fig. 2.

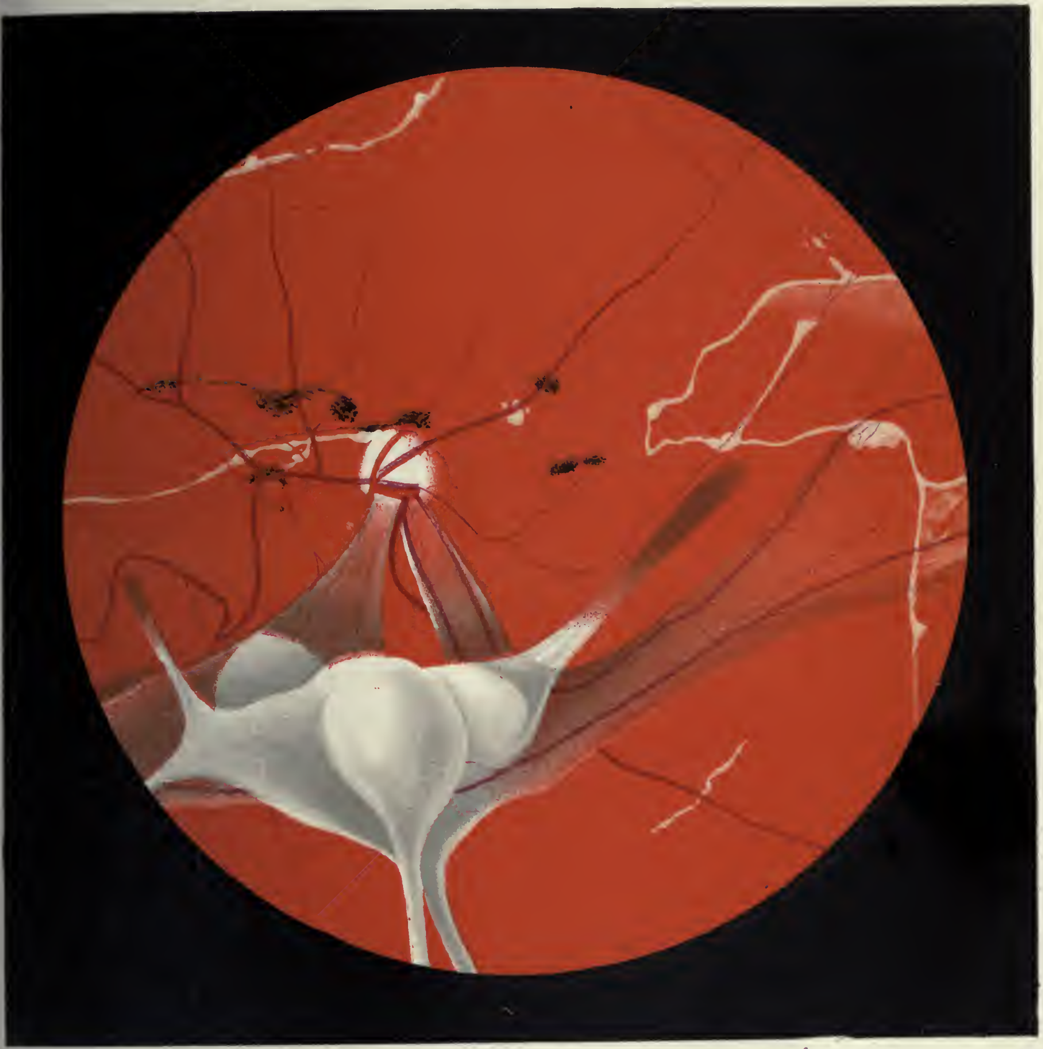
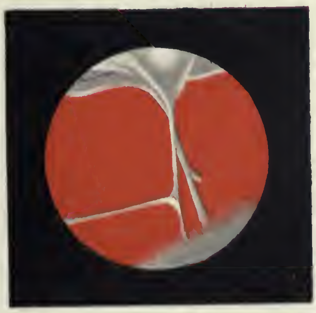


Fig. 1.



Dr. Wintersteiner ad nat. pinx.

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Similar appearances are seen after detachment of the retina, and Liebreich[1] has pronounced them to be coagulates, precipitated from subretinal effusions, whilst Dimmer looks at them as developments of connective tissue in the retina, and Braun,[2] as atrophy of the pigment epithelium.[3]

Stellwag saw white striæ in the retina after retinitis albuminurica, and Berger in eyes with normal vision. The latter thinks that they are the residue of fœtal neuro-retinitis, or of a retinitis occurring soon after birth. The titles of retino-choroiditis and retinitis given by other authors, indicate that they have inflammatory alterations in mind.

Finally, Speiser[4] urges that the retrovascular striæ originate in hemorrhages in the external layers of the retina.

I would also advance another possibility. The striæ are generally arranged radiatingly, and they seem as if passing over elevations in the retina. If now we recall the developmental history of the eye, we remember that the retina in its development often lies in folds, of which we see abundant traces in the fœtal eye as well as in the new-born. It is only when the globe has increased in size with the growth of the child, that these retinal folds disappear. The idea therefore suggests itself, that all the retinal striæ so far observed have some unknown connection with the embryonal folds of the retina.

Other arguments in favor of this origin of the striæ is that no one has so far seen their beginning or development. Onisi alone,[5] after looking at a patient several months later, saw striæ that he thought he had not seen before, but he would not say that they may not have been present at the first examination. Neither have any inflammatory symptoms ever been reported by the patients themselves. They accidentally discovered loss of sight in one eye. Additionally in my case, the striæ, the abnormal vascular distribution, and vitreous opacities insist on a congenital explanation. Finally, Berger and Michel report similar cases, in which the conditions must have originated at an extremely early age, even if they were not congenital.

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[1] *V. Graefe's Archiv*, ii., p. 256.

[2] *Beitraege z. Augenhlkde.*, 1893, Heft, xii., p. 30.

[3] See also a paper in this number of these ARCHIVES by Dr. W. A. Holden.

[4] *Beitraege z. Augenhlkde.*, Von Millinger, Basel, 1893.

[5] *Mittheilungen a. d. Augenklinik. Tuebingen*, ii., 3, p. 377.

A CASE OF DOUBLE COLOBOMA OF THE CHOROID.

By C. A. VEASEY, M.D., Philadelphia, Penn.

(*With an ophthalmoscopic drawing in the text.*)

THE following case of coloboma of the choroid, which seems to possess some points of interest peculiar to itself, presented the following history :

A. S., female, aged twenty-four, came to me for the treatment of a variety of symptoms which she attributed to her eyes. For several years she had had frequent frontal and occipital headaches accompanied by nausea, and occasionally by vomiting, always aggravated by doing any near work. The headache was very severe, and the patient stated that almost every week she was obliged to spend one day in bed on account of it—usually a day in the latter part of the week. There were also present symptoms of accommodative asthenopia and ciliary pain. She had been wearing sphero-cylindrical lenses for three years, with relief of the symptoms for one year after they were obtained. The family history was good. Both her parents and herself denied having had any specific lesion. The general health was very good excepting the disturbance arising from the ocular symptoms. The vision and accommodative power were as follows :

O.D.V. $\frac{5}{7.5}$ Jäger, No. 2——3″–28″
O.S.V. $\frac{5}{15}$ Jäger, No. 2——5″–10″

After she obtained her correction, which was as follows ;

O.D. + S. .75 D ⊃ + C. .75 D. ax. 90°
O.S. + S. 1.25 D. ⊃ + C. 1.75 D. ax. 90°

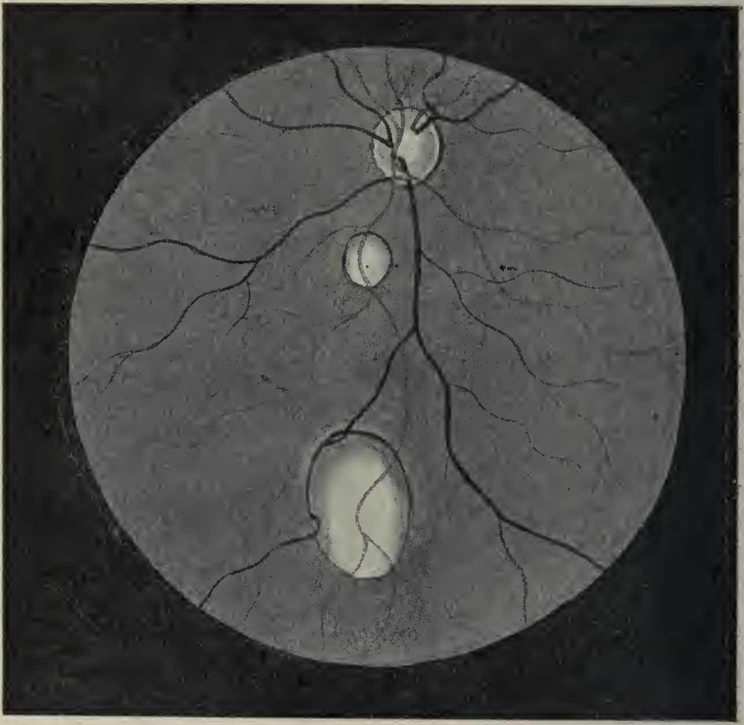
the vision was for the right eye $\frac{5}{5}$ full, for the left eye $\frac{5}{5}$ partial, the best working distance being 16″.

The tests for muscular balance showed no trouble in this direction. Both pupils were normal, reacting quickly to light, convergence, and accommodation. The tension was also normal and the ophthalmoscopic changes were as follows :

O.D.—Media clear, disc oval, deep central physiological cup, absorbing conus to the nasal side of disc, cilio-retinal vessel to the temporal side about the junction of the upper and lower quadrants, choroidal ring all around, no macular disturbance.

O.S.—Media clear, oval disc, deep central physiological cup, choroidal ring all around, veins slightly fuller than normal, but blood apparently not darker than usual. About the distance of two thirds of the diameter of the disc below it and slightly to the nasal side, is a small circular coloboma of the choroid, greenish-white in color, which is about two thirds as large as the disc itself. The margin is pigmented all around while the bottom shows numerous elevations and depressions, presenting an opalescent appearance, and having on its surface two scleral vessels, the one to the temporal side sending off a branch. The depth of the coloboma varies from 1 to 2.50 D. in different parts. Passing over it from above downward is the inferior nasal artery. The surrounding choroid is in a perfectly healthy condition.

About the distance of two diameters of the disc below the coloboma just described is a second, oval in shape and much larger than the former, measuring, as nearly as could be ascertained, about 1½ × 2½ diameters of the disc and being slightly narrower in the lower than in the upper half. The margin of this also is pigmented, much more heavily in the upper than in any other part, and the color, as in the first, is of a dirty greenish-white. The coloboma, in the upper outer portion and in the lower inner portion, slopes gradually from the margin towards the centre, while in the other parts the edge is more abrupt. The bottom presents numerous small elevations and depressions and has an opalescent appearance like the first, the depth varying from 2 to 5 D. The inferior nasal artery that passes over the upper coloboma from above downward continues its downward course, passing beneath a branch of the inferior temporal vein and crossing the lower coloboma in a curve, sending off a branch just before it reaches the lower margin. The branch of the inferior temporal vein just mentioned passes over the edge of the coloboma above, dipping beneath it so as to be entirely lost from view, and then, reappearing near the middle of the outer edge, passes downward and outward. Coming from beneath the upper outer margin is a scleral vessel which sends two branches downward, one to the temporal and one to the nasal side, both being lost under the margin. Above and to the nasal side of the lower coloboma the choroid is distinctly thinner, though in all other directions it appears to be in a healthy condition. The accompanying illustration made from a water-color sketch by Mr. T. C. Lander of this city, shows very well the condition of the fundus.



The field of vision shows a scotoma for form and colors of the exact shape of the smaller coloboma, the larger one being so far in the periphery that it is indicated in the field only by a curve in the upper edge.

The defects are believed to be congenital, an examination by my friend, Dr. Edward Jackson, over three years ago revealing practically the same condition. No other congenital defects could be found in the patient, and an examination of the father and a sister showed nothing more abnormal than low refractive errors.

The case presents the following points of interest:

There are two separate colobomata of the choroid with a wide area of intervening healthy choroidal tissue, in which the field of vision is perfectly normal. Cases have been recorded in which a large coloboma was divided into different parts, as, for example, the case of Hirschberg, quoted by Bock[1] in his excellent monograph, where the coloboma was divided into three parts, while in the middle ran a thin strip of choroidal tissue; or Hoffman's[2] case, in which there was an intervening space of healthy choroidal tissue, but which was no wider than two thirds the diameter of the small coloboma, the latter being about half the size of the disc; but the author has been unable to find any case recorded in which the colobomata were so widely separated, and in which so much of the intervening choroid was in a healthy condition—in the present case the whole space, with the exception of a slight thinning above the lower coloboma, being normal.

The shape of the defects is of interest, the upper being circular and the lower oval. The visual field shows a scotoma corresponding in shape to the upper coloboma, and a curve in its upper margin corresponding to the margin of the lower coloboma. This agrees with the observations of Schmidt-Rimpler[3] and De Wecker,[4] though Johnson[5] claims that it is very seldom that the field shows a defect even slightly resembling the outline of the defect in the choroid.

It is also of interest to note that the choroid below the lower coloboma is in a normal condition. The defect is supposed to be due to the non-closure of the ocular fissure, but why it should have closed in certain parts and not in others, or why, if it were entirely closed, it separated above and below, leaving so wide a space perfectly healthy in almost the entire extent, is only a matter of conjecture.

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[1] Bock, *Die Angeborenen Kolobome des Augapfels*, p. 95.

[2] *Graefe-Saemisch Augenheilkunde*, p. 73

[3] Schmidt-Rimpler, *Graefe's Arch.*, xxiii, 4, p. 176.

[4] De Wecker et Landolt, *Traité Complet. d'Oph.*, vol. ii., p. 515.

[5] Johnson, *Arch. of Oph.*, 1890, p. 28.

DIABETIC RETINITIS.

By OSCAR DODD, M.D.,

INSTRUCTOR IN EYE AND EAR DISEASES, COLLEGE OF PHYSICIANS AND SURGEONS; ASSISTANT SURGEON AT THE ILL. CHARITABLE EYE AND EAR INFIRMARY, CHICAGO, ILL.

ALTHOUGH the existence of diabetic retinitis has been known for a long time, the literature on the subject is very meagre and unsatisfactory. The text-books on general medicine many of them do not mention the possibility of its occurrence, and the works upon the eye only give short statements that it may exist, but no satisfactory explanation of its symptoms. Having seen three cases of the disease within about a year, I think the scattered condition of the literature is apology enough for gathering the histories of all the cases of pure diabetic retinitis reported in literature and gleaning from them the characteristics, if there be any, that are indicative of the disease.

Diabetes being an infrequent disease and retinitis one of its less common complications, we have only a few cases reported, as diabetes is frequently complicated in its later stages (when retinitis most frequently occurs) by albuminuria. This has been considered by some as the cause for the retinitis. For this reason I have excluded from the summary all such cases, except one in which a thorough post-mortem examination had been made, and one of my own cases.

The more frequent complications, as weakness of accommodation, transient amblyopia, and cataract, had been known long before Edward Jaeger published the first report of a case of diabetic retinitis in 1856, a representation of which is given in his ophthalmoscopic atlas (Fig. 64). This was followed by two cases reported by Desmarres in 1858. Although plenty of sugar but no albumen was found in the urine in these cases, the view was still held that possibly there had been a previous albuminuria which was absent at the time of examination. That diabetes alone could cause a retinitis was subsequently shown by H. D. Noyes in 1869, who carefully followed the history of a case of diabetic retinitis and found that while the eye symptoms bore a close resemblance to an albuminuric retinitis, there was no albumen present at any time in the urine, but abundance of sugar, and that the normal amount of urea was present.

Leber was able to collect the reports of 19 cases of diabetic retinitis in 1875 including one of his own. The histories of many of these cases were very meagre so that the presence or absence of albumen was only mentioned in 8 cases. In 4 of these there was no albumen, and they have been included in my report. His classical articles in *Graefe's Archiv*, vol. xxi., and the *Graefe-Saemisch Handbuch der Augenheilkunde* have been long considered authority on the subject. Hirschberg reported 24 cases in his articles in the *Deutsche klin. Woch.*, 1887 and 1890, 15 of which were pure diabetes. Lagrange reports from the eye clinic in Bordeaux the histories of 17 cases, 11 being in pure diabetes. Besides these more extensive articles Nettleship and others have reported cases which have been included in this report.

In order to get the ideas of different authors upon the subject, we will briefly consider them here.

Noyes (*Text-Book of Eye Diseases*, 1894), says: " Retinitis glycosurica is much less frequent than retinitis albuminurica. The features of both are to a great extent identical. The bright yellow patches are less liable to affect the macula; perhaps they lie more frequently between the disc and the macula. They are more nodular and irregular than in the albuminuric form." He then gives a description of the case reported in 1869.

Berry (*Text-Book of Ophth.*, 1889) states: " Retinitis occurs in some cases of diabetes, mostly those of severe type. The form is not characteristic; it resembles albuminuric retinitis, but is accompanied by fewer changes in the region of the macula. Opacities in the vitreous are frequent."

DeWecker & Masselon (*Manuel d'Ophtal.*, 1889,) say: "Most cases of retinitis said to be diabetic occur in diabetic albuminuria; in reality true diabetic retinitis is very rare. Diabetic retinitis consists of simple hemorrhages alone or mixed with foci of degeneration; it is sometimes accompanied by large hemorrhages into the vitreous or coats of the optic nerve."

Juler (*Handbook of Ophthal. Science*, 1893) says: "Diabetic retinitis is very rare, and is so similar to the albuminuric form that it is almost impossible to distinguish the one from the other by means of the ophthalmoscope alone. The white spots in the macular region are large and irregular in outline in this affection, and they have no tendency to be radially arranged around the yellow spot. Hemorrhages occur in all parts of the fundus; the extravasations appear either in the form of dots or as irregular patches and lines. In the early stage papillitis is not a marked sign, but later on it becomes very prominent. The disease tends to progress; new vascular formations appear in the vitreous, and the latter becomes crowded with membranes and other opacities; lastly the retina becomes detached, with complete loss of vision. The retinitis is symmetrical."

Fuchs (*Lehrbuch der Augenheil.*, 1889) only mentions the disease to say that it is very rare, and that the changes in the retina are due to the altered condition of the blood.

Leber, in summing up his article in *Graefe's Arch.* (vol. xxi.), says: "The form of the retinal changes offers little that is characteristic by which we could judge that diabetes is the cause of the trouble. In many cases there are only retinal hemorrhages to which whitish spots of degeneration are added later, frequently accompanied by vitreous opacities. In other cases there is hemorrhagic retinitis as in disease of the heart or blood-vessels, in others whitish spots and hemorrhages which cannot be distinguished from albuminuric retinitis. The spots are small ($\frac{1}{4}$–$\frac{1}{2}$ O D, sometimes 2 O D), never run together, and are scattered over a large part of the fundus."

Schweigger says that all forms of eye disease are seen with diabetes, but none that bear the same relation to it, as for example retinitis albuminurica to Bright's disease or retinitis syphilitica to lues.

In opposition to this view is that of Hirschberg, who has grouped the cases of diabetic retinitis into three principal classes.

1. A characteristic inflammation of the centre of the retina with small bright spots and usually with small hemorrhages (retinitis centralis punctata diabetica).
2. Hemorrhages in the retina with the following inflammatory changes and degeneration (ret. hemorrhagica diabetica).
3. Rare forms of retinal inflammation and degeneration, the connection of which with the fundamental disease must be more fully proved. (He has seen three cases of retinal pigmentation in diabetes with contraction of the visual field and night blindness similar to retinitis pigmentosa.)

The first form he considers indicative of diabetes, and believes it is easily diagnosed from albuminuric retinitis which it most closely resembles.

Lagrange, reporting the cases from the ophthalmic clinic at Bordeaux, says of the disease: "Diabetic retinitis nearly always presents hemorrhages which may be large or small. It is usually easy to distinguish from the cardiac and syphilitic forms, but not so from the albuminuric." He then gives the table of differential diagnosis between the two diseases prepared by Badal, and which will be given below.

Its frequency cannot even approximately be determined as there are no incident statistics. In the ophthalmic clinic at Bordeaux among 20,000 eye cases there were 52 cases of diabetes. Of this number 17 had retinitis, 11 being in pure diabetes, and included in my list.

Galezowoski reported 27 cases with retinal changes, among 144 diabetic eye cases (19 per cent.), but gave no detailed description of them. This was much less than the number of cataracts of which there were 46 (31 per cent.). Hirschberg thinks that eye changes due to diabetes are much more frequent than is generally supposed, and says that retinal changes are probably present in all cases of long standing.

The age at which diabetic retinitis occurs is generally an advanced one, most of the cases occurring after forty-five years of age. The youngest case, that reported by Culbertson, aged six years, was an exception, as the diabetes was secondary to malaria, which may have produced the grave changes in the blood necessary to the production of the retinitis. The reason for its occurring at such an advanced age may be due to the changes normally occurring at this time of life in the blood-vessels, but it is more likely due to the fact that the disease takes a much more protracted course at an advanced age, sometimes lasting for a great many years. Retinitis only occurring at a late stage of the disease would be more apt to occur in such cases where the changes in the blood-vessels had an opportunity to develop.

As to the form of the retinal changes present, we have several types. The one most frequently met with is that described by Hirschberg as retinitis centralis punctata diabetica. The changes are somewhat differently described by the various authors, but they agree in all the essential points. Lagrange described some of the cases under the name of retino-choroiditis, but expressly stated that there were no pigmentary changes.

The changes consisted of small bright glistening spots of degeneration interspersed with minute hemorrhages principally in the central part of the retina. The spots are usually small and rarely exceed ¼–½ the size of the optic disc. In some of the cases, however, there are a few larger ones, but these are surrounded by a number of smaller patches or punctate spots. The spots vary much in shape, being usually nodular and irregular in outline, seldom stellate around the macula as in albuminuric retinitis. They are generally grouped between the disc and macula or near the macula, sometimes in the form of a semicircle or circle at some distance from it. De Wecker reports a case of this kind in his article on retinitis circinata (*Arch. d'Opht.*, January, 1894), which afterwards proved to be diabetic.

The color of the spots varies from a dull to a glistening, brilliant white or yellowish-white. The variation in the color is undoubtedly due to the stage of the disease at which they are seen. These changes are accompanied by minute hemorrhages in most of the cases, which may be absorbed or give place later to foci of degeneration. Besides these changes in the central part of the retina, there may be small spots and hemorrhages to the nasal side of the disc or along the temporal vessels. The spots never run together, covering a large part of fundus, as they sometimes do in albuminuric retinitis.

In another class of cases very similar to these the whitish patches and hemorrhages are scattered over the whole fundus. The patches are larger in size (about that of O D) and are either uniform or interspersed with hemorrhages. De Wecker and Masselon give a good illustration of this class of cases in their *Text book of Ophthalmology*.

Œdema and active changes in the retina and optic nerve are rarely present although occurring so frequently in albuminuric retinitis. The vessels are usually of normal size and contour. This is one of the chief distinguishing features between diabetic and albuminuric retinitis.

Embolism of the retinal artery was present in two cases and thrombosis of the central vein in one case. These changes undoubtedly occurred in cases accompanied by endocarditis, which is so frequently present in the later stages of diabetes (Purdy). That there is a tendency to occlusion in the veins has been shown by the pathological examination made by J. Michel in which he found a partial occlusion of the central vein by a deposit on the inner wall.

The frequency of opacities and hemorrhages in the vitreous has not proved so great as would be supposed from literature, several authors mentioning it as a characteristic of the disease. It was present only seven times among the cases reported. In cases of central punctate retinitis they were very slight when present and the source could not be traced. In the hemorrhagic form they were more abundant and seriously affected vision. Their source could not be positively traced, and it was not known whether they came from the retina or from the vessels of the ciliary body.

The hemorrhagic form was less frequent than that with foci of degeneration. Hirschberg has divided it into four kinds: (1) small punctate hemorrhages, (2) larger hemorrhages accompanied by opacities of the vitreous, (3) hemorrhagic infarction of the retina, (4) hemorrhagic glaucoma. We find the first kind to be most frequent and the least dangerous. The only cases reported as fully recovering were of this variety, but most of the cases later develop foci of degeneration in the spots where hemorrhages were and pass into the mixed form. It is rarely discovered in its early stages except by accident, as the disturbance of vision is very slight, sometimes being only an excessive sensitiveness to light, or flickering before the eyes. Seven cases of this kind were reported, one of which fully recovered. The second form is more serious, as the hemorrhages affect the vision, especially if they break into the vitreous. There is always danger of its developing into hemorrhagic glaucoma. Hemorrhagic infarction of the retina is rare, and the prognosis as to life of the patient is very serious. The only case occurring in pure diabetes was Case 14, and the patient died one year later. Only one case of hemorrhagic glaucoma was reported in pure diabetes, (Case 7), and this occurred after an iritis, but Hirschberg had three cases and Lagrange one, occurring when the diabetes was complicated by an albuminuria. The extensive vascular changes make these cases hopeless.

The *subjective symptoms* of this disease are very slight in the cases with minute hemorrhages and punctate spots in the centre of the retina. The retinitis may be accompanied or preceded by the diabetic amblyopia, which, according to Bouchardot is present in $\frac{1}{5}$ of all diabetic cases and frequently occurs without any fundus change. Most of the cases complain only of a dimness of the vision, especially for near work, and are seen only upon their applying for glasses to correct the defect. There may also be a sensitiveness of the eyes to light and a flickering before the eyes at an early stage of the trouble, due to an irritation of the retina. When the change affects the macula itself or there are large hemorrhages into the vitreous the visual disturbance is profound.

Of the *conditions following diabetic retinitis* the most frequent is atrophy of the optic nerve. Lagrange thinks that in almost every case the optic nerve is in a state of atrophy if it is not already completely sclerosed. It is quite different from that occurring with albuminuric retinitis which is preceded by a neuritis. The atrophy in diabetes is primary, due to the disturbance in nutrition or pressure from a hemorrhage into the sheath of the nerve.

The *pathology of diabetes itself* is not established, and we probably include several conditions, differing in their pathology and pathological anatomy. The changes which would affect the eye principally are those of the blood-vessels and nutrition. In the *Traité de Médecine*, 1893, vol. i., we find the following: "Endocarditis is present usually upon the auricular face of the mitral valve. The arteries of the brain and retina are frequently affected with fatty or atheromatous degeneration (Dupuytren, Charcot, Lecherché). Retinal hemorrhages are frequent, either due to the dyscrasia of the blood, to the fragility of the vessels, or to a simple atrophy, general or partial, of the retina and optic nerve."

Purdy (Diabetes 1889) states: "In most cases of diabetes there is an increased vascular tension as in chronic Bright's disease. It would seem that an extra demand is made upon the heart with primary hypertrophy of the left ventricle."

Osler (*Practice of Med.*, 1892) says: "The kidneys are sometimes fatty and show a hyaline change in the tubular epithelium particularly in the descending loop of Henle. This change also occurs in the capillaries. Endocarditis is very rare. The heart shows no characteristic changes."

Ludwig Mauthner, in an article upon the affections of the eyes in diabetes (*La France Médicale*, 1893), gives four means by which it affects the eye:

1. Subtraction of water from the tissue, with the presence of glucose (producing cataract, etc.).
2. Diminution of the resistance of the tissues, due to the general affection of the nutrition.
3. The presence of a toxic substance in the blood which produces the degeneration and inflammation.

4. Cachectic troubles. Affections of the nutrition would diminish the resistance of the blood-vessels so as to allow transudation of the blood corpuscles from the blood-vessels into the retinal tissues, but it is to the toxic substance in the blood acting upon the blood-vessels, especially the capillaries, to which most of the changes are due. These changes favor hemorrhages from the smaller vessels and the *localized* degenerations such as are possible with a system of terminal arteries without anastomoses. The optic atrophy, so frequently occurring with the retinitis, or alone, is due to the hemorrhages into the sheath of the nerve.

Of the *pathology of the ocular changes* only a few examinations have been made. The most extensive was the case (No. 8) reported by Stephen Mackenzie, the examination of the eye being made by Mr. Nettleship. It was briefly as follows:

There was a hyaline degeneration of the intima of the blood-vessels, especially the smaller ones, of the kidneys, spleen, liver, and some of the smaller vessels of the brain. This hyaline thickening of the walls was not evenly distributed but formed bulging masses extending into the lumen of the vessel. The epithelium of the secreting tubes in the kidney was granular and swollen. In the eye there was thickening of the retina in all its layers, but most in the nerve fibre layer where there were numerous varicose swellings. The disc was slightly swollen and the meshes formed by the separated nerve fibres were partly empty and partly filled by swellings similar to those in the retina. The swelling in other layers appeared to be the result of a chronic œdema with hypertrophy of the connective-tissue framework. The bacillary layer was wanting and its place was occupied in some parts by an albuminuric effusion. Here and there in the intergranular and molecular layers were round spaces (microscopical cysts) filled with faintly refracting globules (albuminous). The limitans interna was much thickened in parts and thrown into abrupt folds. No hemorrhages in retina. Vitreous in left eye contained a large quantity of blood which appeared to come from different spots in a gradual manner, but its source could not be traced. The retinal arteries were greatly changed. Thickenings were present between the epithelium and the fenestrated membrane affecting only one side of the vessel and contracting the lumen very much. The changes affected all branches of the central artery but varied greatly in degree in different portions. As a rule the relative thickening of the coats was much greater in the very minute arteries than in those of larger size. At an early period the new material was more or less distinctly corpuscular and its inmost portion sometimes appeared composed of superimposed layers of endothelia. In many places where the disease was apparently of older date the appearance is best described as partly hyaline and partly fibrous, the hyaline changes being obvious in the smallest vessels. The muscular coat was not generally affected. The veins were merely distended, the coats not being altered. The capillaries of all parts were distended. In the retina were minute capillary aneurisms not very abundant but occurring in groups of about half a dozen. They were globular, apparently communicating with only one side of the vessel, never fusiform dilatations. They contained some reddish-brown granules and there were some granules just external to them suggesting that the hemorrhage into the vitreous may have been due to the rupture of a number of these minute sacs. The choroid was greatly thickened owing to distention of the vessels and infiltration.

In case No. 11, reported by Mr. Nettleship a microscopical examination of the eye was made by Mr. Lawford. There was thickening and puckering of the central artery, with the lumen almost obliterated. Veins thickened. Hyaline thickening of the vessels of choroid and some of the retinal vessels. Some large masses of waxy-looking granular material were observed in both granular layers, with smaller-sized ones in nerve-fibre layer. Capillary aneurisms. It was probably a true inflammation commencing around the vessels and accompanied by an exudate of inflammatory material into the retinal substance which pressed upon and destroyed the nervous tissue and finally underwent fatty degeneration.

In Michel's cases (No. 9) there was a double parenchymatous nephritis in stage of infiltration, hyperaemia of the brain, and a large caseous and ulcerating exudate in right lung. Microscopical examination of the eyes showed an infiltration of blood corpuscles between the nerve-fibre bundles and the separated nerve fibres of the right optic disc. There was partial closure of the lumen of the vena centralis for only a short distance ($\frac{1}{2}$ *mm*). The lumen was about $\frac{2}{3}$ filled by a mass firmly attached to the wall of the vessel. It consisted of a finely striated tissue, containing endothelial and lymph cells, with a few blood corpuscles.

Besides these, Galezowski examined case No. 6, where secondary glaucoma had developed. The sclera was bulged in numerous places, but there was no change in its structure. Fibrous exudate in choroid. Retina was detached and the eye filled with blood.

We see from these cases that the important changes are in the blood-vessels. These affect principally the small vessels, and are not so extensive as in albuminuric retinitis. The partial closure of the vessels tends to rupture, producing the small hemorrhages which are so common in this disease, and predominate over the degeneration which is the characteristic change in albuminuric retinitis. If there is anything in the pathology of the disease distinguishing it from other forms of retinitis it is the peculiar changes taking place in the small arteries and capillaries. In other forms of retinitis we have changes in the blood-vessels, but they are equally as great or predominate in the large vessels.

The *differential diagnosis* of this disease from others which resemble it is one of the principal objects of this paper. Of the two principal forms of the disease one is characterized by spots of degeneration alone or mixed with hemorrhagic spots, and the other by hemorrhages alone. The former is the most frequent and the most difficult to distinguish from albuminuric retinitis, which it closely resembles. Most authors say that it differs from it in some particulars, but give no definite points on which to differentiate. Lagrange, in his article, quotes the table prepared by Badal for the differential diagnosis between the two diseases.

Diabetic Retinitis.	*Albuminuric Retinitis.*
1. Marked tendency to atrophy of the optic nerve.	1. Less tendency to atrophy, which occurs especially at a late stage.
2. Multiple hemorrhages, round and disseminated.	2. Hemorrhages equally multiple but elongated, and occupy especially the posterior layers.
3. Alterations diffuse.	3. Affect principally the circumpapillary and macular regions.
4. Apoplexies do not last to a late stage.	4. Last throughout life.
5. Small disseminated spots, with some exudate.	5. Whitish fatty spots, with infiltrations.
6. Color sense, nil.	6. Persists a long time.

Galezowski says that retinitis glycosurica presents, besides hemorrhages and fibrinous exudate, an alteration of the papilla, conducive, later, to atrophy. An optic neuritis is not found here as in albuminuric retinitis. Albuminuric and syphilitic retinitis are almost always binocular, while glycosuric retinitis is frequently monocular.

Noyes, as already quoted, says that the spots are more nodular and irregular than in the albuminuric form.

The principal differences which I should give as the distinguishing features between the diseases, as gathered from the cases quoted, I will briefly tabulate as follows:

Diabetic Retinitis.	*Albuminuric Retinitis.*
1. Groups of bright glancing spots in retina, irregular in outline, usually in central part, but frequently affecting whole of fundus.	1. At first a group of bright bluish-white spots in centre of retina, often forming stellate patch about the macula.
2. If the spots are larger there still exist small dots and lines, and they never run together.	2. Spots may run together and involve all of central part of retina.
3. Arteries and veins not much changed in appearance.	3. Arteries narrowed, veins large and irregular.
4. Optic nerve either not affected or atrophic.	4. Optic nerve swollen and outline indistinct.
5. Retina not diffusely affected.	5. Retina infiltrated.

The points of diagnosis are not well marked in all cases, and in some the appearance is that of the albuminuric form, with the tortuous vessels, swollen infiltrated nerve and retina, and the grouping of the spots of degeneration about the macula. Under these circumstances nothing but an examination of the urine would allow one to conclude as to the cause.

The purely hemorrhagic form is less difficult to diagnose, for, with the small punctate hemorrhages, it presents a unique appearance, so different from the other forms of hemorrhagic retinitis, that it should suggest at least the presence of diabetes.

We have already seen that most cases of retinitis occur at an advanced stage of the diabetes, after other symptoms have called attention to the disease. In some of the cases, however, the notice of the patient was first called to their disease by the oculist, after examining the eyes. (See cases Nos. 29 and 46.)

As to the *prognosis* regarding the life of the patient, the occurrence of the retinal changes has been considered a grave symptom. Yet in most of the cases where the histories were followed the patients were alive several years after, and in some the diabetes was entirely cured.

As to the *treatment* of the disease there is little indication to do anything else than combat the constitutional dyscrasia by proper diet. In two of my cases the ocular symptoms appeared a few months after discarding the antidiabetic diet prescribed and returning to their old ways of living. In one of them the vision improved upon returning to a strict diet without the starchy foods. Leber recommended the administration of carbolic acid, but other writers have found that while it may have a beneficial effect in diabetic amblyopia, it does not influence the course of the retinitis.

Galezowski considers the disease curable. "At the beginning an antiphlogistic treatment (bleeding behind the ear, etc.) should be used. After the congestive stage terminates, resolving remedies (such as eserine, injections of pilocarpine, collyria of iodide of soda and potassium 1 per cent.) should be combined with the general antidiabetic treatment.

LIST OF CASES.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
1	F	46	3 yrs.		Opacities in vitreous. Large and small bright spots scattered over whole of fundus, mixed with retinal hemorrhages partly old and partly fresh.	Desmarres, *Traité des mal. des yeux.* 1858.
2	M	55	5 yrs.	4 mo.	Several large white patches (1½ to 2 times O D) surrounded by a number of smaller white patches and hemorrhages.	Desmarres, *Traité des mal. des yeux*, 1858.
3	F	60	3 yrs.		Had iritis both eyes a few months ago. Whitish glistening spots in the retina between the O D and macula forming circle around macula. They look like fatty degeneration and similar to albuminuric retinitis. A few hemorrhages. Slight haziness of vitreous.	H. D. Noyes, *Trans. Am. Ophth. Soc.* 1869.
4	F	29	5 yrs.	14 days	Diffuse opacity of vitreous. Papilla grayish white and not clearly demarcated. Retina hazy in central part. Arteries thin, veins large. A large number of small round and irregular hemorrhages. No whitish spots, but some appeared later in places where hemorrhages were.	Haltenhoff, *Zehender's klin. Mon.* 1873.
5					Upper nasal branch of central artery is obliterated and degenerated to a whitish string. Numerous extravasations of blood and some whitish spots.	Galezowski, *Recueil d'Ophtal.* 1873.
6					Hemorrhages and whitish extravasations in retina. Later iritis serosa and acute glaucoma. Microscopical examination made. (See page 216).	Galezowski *Receuil d'Ophtal.* 1873.
7	F		2 yrs.		Fundus is opaque showing œdema of the retina or an exudate between memb. limitans interna and hyaloid membrane. There are whitish spots of exudate and striped hemorrhagic extravasations along the arteries in macular region. Hemorrhages were absorbed and exudate disappeared.	*Umé Arch. méd. Belges* 1875.
8	F	52	1 yr.	3 mo.	Has two large floating films in right eye from blood clot. Vitreous turbid. An opaque linear projection into the vitreous was seen, on which retinal vessels were visible and to which one of the filmy floating webs is attached. Left eye vitreous has the appearance of a diffuse hemorrhage. Only a glistening white crescentic spot was seen near disc but not in connection with it. Died three months later. P M examination made. (See page 214).	Stephen Mackenzie, *Ophth. Hosp. Rep.*, vol. ix., 1877.
9	M	21	1 yr.		Right eye showed a large round hemorrhage upon O D. Much emaciated. Died soon after examination. P M examination made. (See page 216).	Michel, *Deutsche Arch. f. klin. Med.* 1878.
10	F	6		4 wks.	Small white spots at outer side of O D near macula similar to albuminuric retinitis. Outlines of disc obscure. Arteries and veins slightly smaller than normal and papillæ red on outer side. Media clear. No hemorrhages. Under strict diet and treatment recovered. Right eye ended in atrophy of retina and optic nerve. Diabetes was secondary to malaria.	Culbertson, *Detroit Lancet*, 1882.
11	F	63	18 yrs.	Rt. eye 2 yrs. Lft. eye 1 day	Right cholesterine crystals in vitreous. Large number of yellowish white spots of different sizes in retina in region of macula. No haze of retina or hemorrhages. Spots look old. L E yellowish patches not so abundant as in right. Diffuse haze of central part of retina and O D as in embolism of retinal artery. Main lower branch of artery is obliterated by plug at its origin, near O D. Numerous small hemorrhages up to periphery. Has slight trace of albumin in urine. Died six months later from gangrene. P M examination made. (See page 215).	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1882.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
12	M	50		1 yr.	Has large number of spots and patches of whitish degeneration in central part of retina, similar to albuminuria retintitis but not grouped the same. They are more scattered and not spoke-shaped. Spots lie beneath blood-vessels never above them. They are somewhat yellower than albuminuric retinitis.	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1886.
13	M	56	Health good		L E has small grayish spots in region of macula. R E has hemorrhages near O D.	Hirschberg, *Deutsche med. Woch.* 1887.
14	F	71			Has hemorrhagic infarction and complete plugging of central vein. Numerous hemorrhages over whole of fundus. Hemorrhages absorbed later leaving spots of degeneration. Died one year later.	Hirschberg, *Deutsche med. Woch.* 1887.
15	M	64	Sev. yrs.		Cat. right eye. Left eye has central retino-choroiditis around macula and presents all the appearance of alb. retinitis.	Lagrange, *Archiv. d'opht.* 1887.
16	F	65	17 yrs.	2 yrs.	Slight infiltration of papilla. Small spots of exudation around papilla with reflection like mother-of-pearl interspersed with small hemorrhages. In several places large plaques of choroidal atrophy. Floating opacities in vitreous.	Lagrange, *Archiv. d'opht.* 1887.
17	F	53	5 yrs.		Right eye œdema of papilla with small hemorrhages around it. Left eye papilla excavated and in state of atrophy without increase of tension.	Lagrange, *Archiv. d'opht.* 1887.
18	M	53	3 yrs.		Small ecchymoses around the macula which disappeared in a few weeks. Central scotoma L E. Vision became normal.	Lagrange, *Archiv. d'opht.* 1887.
19	M	40		1 mo.	Hemorrhagic retinitis characterized by small hemorrhages scattered around papilla. Papilla swollen and irregular in outline. In places the small vessels are covered by the spots in question.	Lagrange, *Archiv. d'opht.* 1887.
20	M	67	3 yrs.		Small hemorrhagic spots in region of macula and upon O D. Until death four years later alternate amelioration and aggravation of the trouble.	Lagrange, *Archiv. d'opht.* 1887.
21	F	62	Sev. yrs.		Left eye nearly blind from numerous hemorrhages in the vitreous. R E central corona of white (mother-of-pearl) spots around macula similar to albuminuric retinitis. Large number of small hemorrhages in posterior part of fundus.	Lagrange, *Archiv. d'opht.* 1887.
22	F	57		2 yrs.	Central retinitis both eyes which has all the appearance of albuminuric retinitis. Exudative corona around papilla interspersed with small hemorrhages. Beginning posterior polar cataract both eyes.	Lagrange, *Archiv. d'opht.* 1887.
23	F	63	Sev. yrs,		Central retinitis with spots and hemorrhages around macula like albuminuric retinitis.	Lagrange, *Archiv. d'opht.* 1887.
24	M	53	7 yrs.		Hemorrhages radiating from papilla both eyes. Whitish spots in region of macula.	Lagrange, *Archiv. d'opht.* 1887.
25	M	51	5 yrs.	Sev. mo.	Left eye vision entirely obliterated. Small hemorrhagic spots irregularly distributed around macula. Optic papilla in stage of atrophy. Floating opacities in vitreous. R E exudative retino-choroiditis in region of macula, small spots of hemorrhage, commencing atrophy of O D.	Lagrange, *Archiv. d'opht.* 1887.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye Trouble.	Condition of Fundus.	Reference.
26	M	48	4 yrs.	2 yrs.	Left eye became blind one year ago from secondary glaucoma after plastic iritis with occluded pupil. Several enlarged blood-vessels in iris. Right eye has retinitis with haziness. Near macula are yellowish, densely opaque spots such as described in Case 12. Retinal veins distended and scattered hemorrhages. Near fovea centralis 2-3 vessels of medium size appear in retina as though they came from choroid. They form a fine plexus extending into the vitreous.	Nettleship, *Trans. of Ophth. Soc. of the U. K.* 1888.
27	M	49	3 yrs.		Hemorrhages and white patches in retina scattered over whole of fundus. Vitreous clear.	James Anderson *Ophth. Rev.* 1889.
28	M	70	1½ yrs.		Opacity of lens. Two large white spots between O D and macula and remains of old hemorrhage.	James Anderson *Ophth. Rev.* 1889.
29	M	45			Health good. Complains of weakness of accommodation. There was one small hemorrhage in central part of left fundus. Two years later there were small indistinct spots and hemorrhages over whole of fundus. Hemorrhages are partly round and partly elongated.	Hirschberg, *Deutsche med. Woch.*, 1890.
30	M	53	6 yrs.	8 days	Complains of recurring attacks supposed to be glaucoma. Has bright spots of degeneration above and at some distance from papilla, also round and pointed hemorrhages.	Hirschberg, *Deutsche med. Woch.*, 1889.
31	F	58	3 yrs.	4 wks.	Large number of bright spots in central part of retina and a few hemorrhages. A single bluish white spot of exudate.	Hirschberg, *Deutsche med. Woch.*, 1890.
32	M	65		2 mos.	There were a dozen small white spots between O D and macula. A small group of such spots a short distance from O D and a single small hemorrhage in the centre of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
33	M	53		3 mos.	Macular side of O D is pale. Delicate white spots (round and striped) like fatty degeneration in centre of retina both eyes.	Hirschberg, *Deutsche med. Woch.*, 1890.
34	M	65	3 yrs.	4 wks.	Right eye has delicate changes. A few small hemorrhages with occasional white spots. L E extensive central opacity of retina with larger bright spots and hemorrhages along the enlarged veins.	Hirschberg, *Deutsche med. Woch.*, 1890.
35	M	63	6 yrs.	1 yr.	Lenticular opacities. Retina has delicate fine opacities along some of the vessels; to the side of this small bright points, small hemorrhages, and one large brown spot from former hemorrhage, in centre of fundus.	Hirschberg, *Deutsche med. Woch.*, 1880.
36	M	68			After cataract extraction examination showed clear media, small hemorrhages, and white spots in centre of retina and around O D.	Hirschberg, *Deutsche med. Woch.*, 1890.
37	M	63		2 yrs.	Became near-sighted two years ago. Lens opaque. Small hemorrhages and glistening white spots in central part of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
38	F	69	3 yrs.		Opacity of lens. Small whitish spots and small hemorrhages here and there in central part of retina.	Hirschberg, *Deutsche med. Woch.*, 1890.
39	M	56	Long time.		L E has group of fine spots in central part of retina near O D. R E has a hemorrhage on O D.	Hirschberg, *Deutsche med. Woch.*, 1890.
40	F	62	8 yrs.		Lens opaque. Whitish opacities in centre of fundus similar to senile retinitis.	Hirschberg, *Deutsche med. Woch.*, 1890.

Case No.	Sex.	Age.	Duration of Diab.	Dur. of Eye trouble.	Condition of Fundus.	Reference.
41	F	48	3 yrs.		Small punctate hemorrhages in retina. L E has had iritis.	Hirschberg, *Deutsche med. Woch.*, 1890.
42	M	43	6 mo.		Four bright sharply defined round spots about 2 to 25 *mm* in size and a few small white ones with hemorrhages were situated outward from macula. Later other hemorrhages appeared and a vessel which ran underneath had a mottled appearance.	Seggel, *Münch. med. Woch.*, 1891.
43	F	64	Sev. yrs.		Peripheral striæ in lenses. Typical diabetic retinitis. Intensely bright white spots arranged around macula in streaks and lines, perhaps approaching the stellate appearance of alb. retinitis more than usual. Other spots of degeneration less marked and finer farther from macula though few extend beyond O D. Hemorrhagic spots scattered among white ones to nasal side of papilla. Papilla and vessels normal.	Fridenwald, *Arch. of Ophth.*, 1891.
44	M	64			Cataract of one eye. The other eye had numerous small blood spots in retina over nasal part of fundus. Vitreous clear. V = $\frac{20}{30}$. Urine 4 per cent. sugar, no albumin. Three months later the fundus was normal; no hemorrhagic spots. One month later a few fresh hemorrhages, and yet five months later a large hemorrhagic spot appeared near papilla. Died two years after first examination.	F. C. Hotz, Report of case kindly furnished me.
45	M	55	5 yrs.	6 mo.	Right eye has medium-sized white patches scattered over whole of fundus. In the left a number of whitish spots are interpersed with hemorrhages. The changes are more recent than in right. Vitreous clear.	Case seen by Author in clinic at Moorfields, London.
46	F	52	4 yrs.	Recent	Has diffuse opacity of vitreous both eyes. In right eye there was a small whitish spot of exudate between the optic disc and macula and one in upper part of fundus with a few small round hemorrhages. Left eye had only one small spot of exudate near macula. Veins large and fundus appeared hyperæmic. V = $\frac{20}{50}$ each eye.	Case seen in Author's practice, Dec. '93.
47	F	62	6 yrs.	6 mo.	R E has a number of brilliant white spots in macular region, one large one ($\frac{1}{2}$ size *od.*) surrounded by a number of small punctate spots. A few hemorrhages along the vessels in the outer part of fundus. L E has a number of small white spots scattered around macular region. Two weeks later the hemorrhages in right eye had disappeared, their place being occupied by whitish spots of degeneration. Three months later the vision was considerably improved but the fundus conditions were similar. Patient has some albumin in urine.	Case referred to Author by Dr. Wilder, Ill. Eye and Ear Inf.

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ALVEOLAR FIBRO-SARCOMA OF THE OPTIC NERVE.[1]

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(*With five figures in the text.*)

I AM indebted to Dr. E. Lopez, of this city, for the pathological specimen which constitutes the subject of this paper. He has furnished me with the following

CLINICAL HISTORY.

On June 25, 1890, Dr. Lopez was consulted at his office by a lady, fifty-five years of age, for a protrusion of the right eyeball this had commenced about a year before, and had since then increased continuously ; there was no pain or inflammatory reaction connected with this and it could not be assigned to any particular cause. *St. Praes :* The right eyeball protruded in a directly forward direction, being almost out of the orbit ; it was immovable ; the pupil was directed upwards and outwards ; it was dilated and did not react to light ; the lens showed an incipient opacity ; there were also floating opacities in the vitreous. The ophthalmoscope revealed an optic neuritis, the margins of the disc were obliterated, the retinal vessels being only visible at some distance from the disc. $T > n$. $H + 4$ D (with ophthalmoscope) $V = 0$. Left eye : Tn. Myopia $- 3$ D. $V = \frac{1}{3}$ (with correction). Only noticeable lesion was an incipient opacity of the lens. *Diagnosis :* Tumor of the optic nerve or its sheaths (RE).

The general health of the patient being good, and in view of her pusillanimity, Dr. Lopez recommended a purely palliative treatment till the progress of the disease should make the patient understand the necessity for operative interference.

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[1] Studied in the laboratory of Dr. C. M. Desvernine.

The patient was again seen by Dr. Lopez six months later; there were now severe neuralgic pains in the whole area supplied by the ophthalmic branch of the fifth nerve; the eyeball was entirely out of the orbit, the lids closing behind it; the conjunctiva was congested, swollen, œdematous; the cornea had sloughed; the projection forward of the eyeball amounted to 2 *cm.*

The next day, aided by Drs. Larios and Silverio, Dr. Lopez, after performing a preliminary enucleation, extirpated a hard cylindrical tumor, which occupied the centre of the orbit. The tumor had produced a dilatation of the optic nerve, which had attained a diameter of 1 *cm*; it extended from the eyeball to the optic foramen.

The patient recovered from the operation, but a year later died with cerebral symptoms; for some time previously she had been idiotic; the left eye had also been blind for some time, showing a total atrophy of the optic nerve.

HISTOLOGICAL EXAMINATION.

I did not receive the pathological specimen till 1891, and through unavoidable circumstances was unable to study it till 1894. The tumor had been cut up into a number of small semicylindrical pieces, it being impossible to determine the topographical relation in which they stood to one another; the eyeball, which had lain all this time in alcohol, was shrivelled up.

On laying the eyeball open by an equatorial section I found that the tumor had extended into the interior of the eye; at the papilla it formed a prominent mushroom-shaped projection, which was raised about 5 *cm* above the surface of the adjacent retina; its passage into the latter was rather abrupt (Fig. 1). The retrobulbar tumor was cylindrical, softish, granular, and was surrounded by a thick fibrous capsule; its diameter was about 1 *cm.*

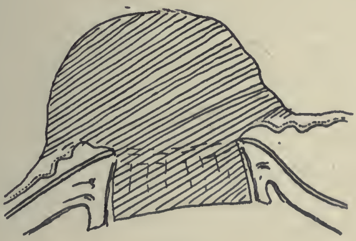

FIG. 1.

Section through optic-nerve entrance, showing the projection of the tumor into the interior of the eye.

MICROSCOPICAL EXAMINATION.

(A) *Retrobulbar Tumor.* This does not present the same structure at all points. In a few sections it is more highly organized. From the thickened and dense dural sheath numerous connective-tissue septa extend into the interior of the tumor, which dividing and uniting with the subdivisions of other septa divide the mass of the neoplasm into a large number of alveoli (Fig. 2), which are filled with closely packed cells; these are mostly spindle-shaped, some, however, are round and others polyhedral. The spindle cells, which constitute the bulk of the tumor, have not everywhere the same appearance; in *some few* of the alveoli they contain more protoplasm and are somewhat wider than is usually the case in an ordinary spindle-celled sarcoma; between these there is a very small amount of intercellular substance. These cells remind one somewhat of endothelial cells, although their appearance is by no means characteristic. The great majority of the alveoli are filled with ordinary sarcoma spindle cells of different size with a moderate admixture of round cells and leucocytes, and between them (within the alveoli) there is a delicate but distinct fibrillar intercellular substance. The fibrous

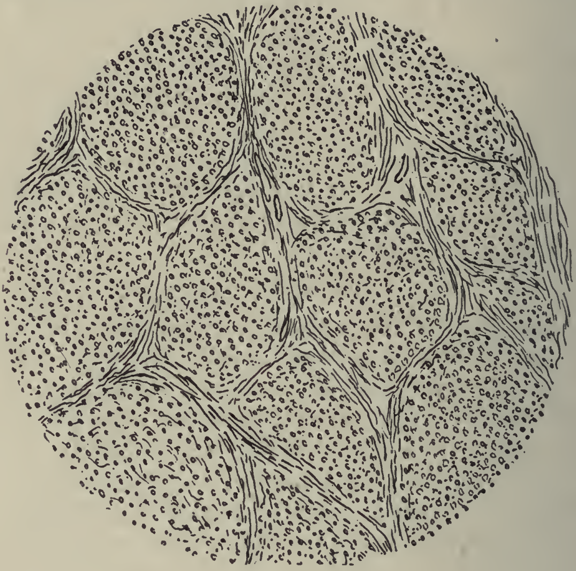

FIG. 2.
Portion of retrobulbar tumor showing alveolar structure.

septa contain a large number of blood-vessels and are rich in cells. There is everywhere an intimate connection between the cells within the alveoli and those constituting the stroma.

In the vast majority of the sections this alveolar structure is absent, the tumor consisting of a dense accumulation of smaller and larger fusiform cells, intermingled with a relatively large proportion of round cells and moderately infiltrated by ordinary white blood corpuscles (Fig. 3). The arrangement of these cells is extremely irregular—at places they are grouped together in bands which cross each other at all possible angles—at places the arrangement follows no particular rule—in the peripheric portions the cells affect a more or less concentric arrangement. There is a delicate intercellular fibrous network which at places is very prominent, reminding us of a young fibroma, and which at places is completely masked by the cells; between these extremes we find the proportion between cells and intercellular substance varying greatly. Blood-vessels are to be found scattered throughout the tumor; their walls are thick. At places the spindle cells are arranged more or less concentrically around them (as seen when the vessels appear cut perpendicularly to their axis) or along the vessel walls, the long axes of the spindles running parallel with that of the vessel (when the vessels appear in longitudinal section), forming sheaths for the vessels; their endothelium is everywhere unaltered.

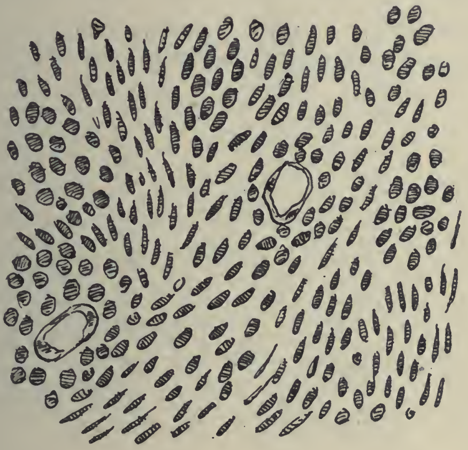

FIG. 3.

Portion of retrobulbar tumor, showing more general atypical fibrosarcomatous structure.

The fibrous capsule of the tumor is formed by the thickened dural sheath, and is made up of dense fibrous tissue. In only one slide is an extension of the neoplasm into the neighboring orbital cellular tissue noticeable—that was at a point of exit of a blood-vessel, the thickened walls of which as well as the immediately surrounding cellular tissue are infiltrated with sarcoma spindles ; this infiltration is limited to the cellular tissue just outside the sheath ; the lumen of the vessel was quite free.[1] Nowhere in this portion of the tumor can any trace of nerve-fibre be found.

In some places the intervaginal space is free, in others no space can be discovered between the thickened dural sheath and the mass of the neoplasm.

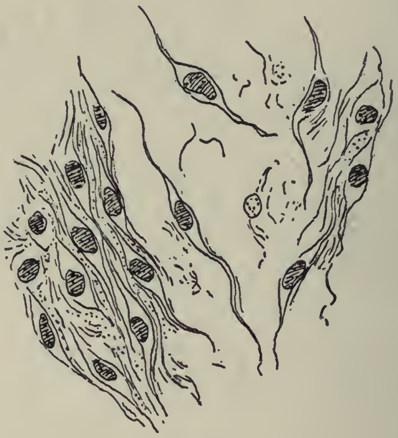

FIG. 4.
Highly magnified section through intraocular tumor.

(*B*) *Intraocular Tumor.*—This portion of the tumor presents a totally different aspect ; the cells are no longer as closely packed together, but wider apart, and between them the cellular interspaces are occupied by granular matter and by an *abundant fibrillar network proceeding from the cell processes.* The cells are mostly spindle-shaped, of which there appear to be two varieties: (*a*) one smaller and wider, (*b*) one longer and more slender ; they are bipolar, with long fibrillar processes, which mingling with those of the neighboring cells form the fibrillar meshwork just mentioned (Fig. 4). There is also an abundant admixture of round cells and of leucocytes. This portion of the tumor occupies almost exclusively the superficial layers of the retina, especially the nerve-fibre layer, from this portion extending secondarily to the deeper-seated; these are quite recognizable and only show some ordinary round-cell infiltration, and here and there some extension of the tumor cells between their normal elements (Fig. 5). This intraocular extension of the tumor is moderately vascular, the blood-vessels showing thickened walls with fusiform cells accumulated around them. At one or two places near the optic-nerve entrance the retina is raised from the choroid by extravasated blood. The extension of the neoplasm into the interior of the eye via the interstitial tissue of the optic nerve is very apparent. At the optic-nerve entrance the intervaginal space is quite free.

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[1] This bears out Dr. Holden's views on the extension and development of sarcoma by a proliferation of the adventitial cells of the vessel walls (ARCH. OF OPHTH., xxii., 3, 289).

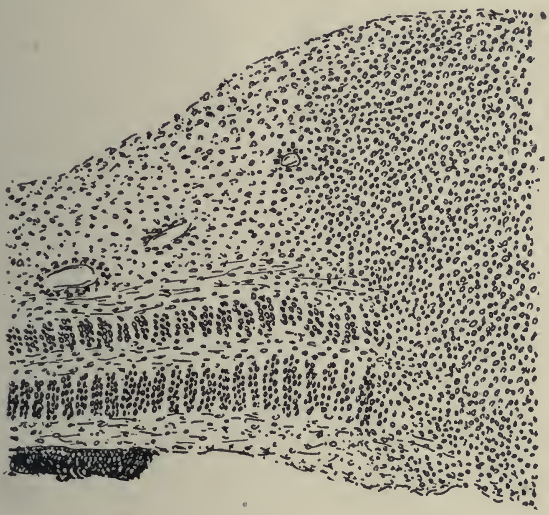

FIG. 5.
Section showing invasion of retina by neoplasm.

The choroid is normal, at least as far as relates to an involvement by the neoplasm.

HISTOLOGICAL CONSIDERATIONS.

It is curious to note the differences in structure presented by this tumor at different points. The great bulk of the neoplasm is evidently a fibro-sarcoma of the small spindle-cell type, in which the structure is as usual atypical; but then we have a portion presenting a curious alveolar structure, the cells within some of these alveoli reminding one in their appearance of endothelial cells. This appearance is as already stated not sufficiently characteristic to warrant a definite conclusion as to their nature. Should they be considered as endothelial cells, the tumor would come under the "endothelial sarcomata" of Birsch-Hirschfeld[1]; but this is a very delicate point to settle, and I will classify the growth as an "alveolar fibro-sarcoma." It is a pity that one cannot determine the relative position of these two portions of the tumor. The still greater alteration in structure presented by the neoplasm on its extension into the interior of the eye, is readily explainable by the different physical and biological conditions under which the cells find themselves (resistance, nutriment, soil, etc.). The spreading apart of the cells, the fibrillar meshwork proceeding from the cell processes and granular substance contained in the cellular interspaces, lead me to consider this portion of the new growth, especially that bordering on the vitreous as undergoing *myxomatous degeneration*; no mucin can be found in the cellular interspaces, but this may possibly be explained by the long sojourn in alcohol. This transition from the more highly organized alveolar sarcoma to the more atypical ordinary variety, and into the still more lowly organized myxosarcoma, is very instructive to follow.

There is another example of this kind reported by Neumann,[2] in which an optic-nerve tumor presented a partly alveolar and partly compact structure, in which there were concentrically arranged collections of fusiform cells. The compact portion consisted of thick fibrils containing sarcoma cells in their meshes; the centre of many of the cells was encrusted; the fusiform cells were endothelial. Neumann calls his tumor a psammoma on account of the cellular encrustations.

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[1] This author describes a variety of tumor often erroneously described as "endothelioma," which occur most frequently in the *membranes of the brain*, and which should be properly considered as sarcoma. They present, in a fibrillar (or myxomatous), more or less cellular stroma, accumulations of endothelial cells. He also mentions the possible occurrence of bands of endothelial cells in sarcomatous tumors of different organs, which proceed apparently from a proliferation of the connective-tissue endothelium or of that of its lymphatics. He considers that these tumors should be denominated "endothelial sarcomata," in view of their preponderating cell growth, these tumors clinically fitting into the "fibro-sarcomata." Birsch-Hirschfeld also seemingly differentiates between the "endothelial alveolar sarcoma" and the endothelioma proper (endothelial cancer).—(*Lehrbuch der alg. path. Anatomie*, Birsch-Hirschfeld, Leipsic, 1889, p. 185).

[2] *Arch. f. Heilk.*, xiii., 310.

GENERAL CONSIDERATIONS.

On looking up the literature of optic-nerve tumors, I find that I have been able to gather together a much larger number of cases than previous compilers. Willemer[1] collected 27 cases, Vossius[2] 34, Jocqs[3] 62, Braunschweig[4] 61, whilst I have managed to collect, either directly or through extracts, reports of 117 (including the one here described). I have arranged these cases in a tabular form at the end of this paper, noting the points of most interest as regards nature, age, duration, operative interference, recurrence, etc. Among these I have included 5 cases which rightly do not belong to the optic-nerve tumors,—*i.e.*, 3 cases of large calcareous concretions in the optic nerve, and 2 cases of aneurism of the central artery of the retina; on the other hand, I have made no mention of Jacobson's case,[5] as here there was no optic-nerve tumor proper. These tumors may be grouped together as follows:

Calcareous concretions	3	*Endothelial sarcoma*	1
Aneurisms of the cent. art. of the ret.	2	Endotheliomata	5
Cysts[6]	2	Gliomata	3
Fibromata	9	Myxo-glioma	1
Myxomata	13	Glio-sarcoma	2
Myxo-fibromata	3	Carcinomata (metastatic)	3
Sarcomata[7]	12	Gumma	1
Fibro-sarcomata	4	Tubercular tumors	4
Myxo-sarcomata	14	Tumors with no or doubtful denomination	33
Alveolar sarcomata	2		
	64		53
Total			117

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[1] *Arch. f. Ophth.*, xxv., 1, 16.
[2] *Arch. f. Ophth.*, xxviii., 3, 33, 283.
[3] *Thèse de Paris*, 1887; remarkably complete work.
[4] *Arch. f. Ophth.*, xxxix., 1.
[5] *Arch. f. Ophth.*, x., 2, 55.
[6] One was a hydatid.
[7] Among these is included Dusausay's angiolithic sarcoma (*Bull. de la Soc. Anat.*, xx., 1875), which possibly would be better included among the endothelial sarcomata.

In these, as in other statistics, one can readily see the immense preponderance of tumors of the connective-tissue type, which here seem to be especially prone to myxomatous degeneration. As regards the special variety of sarcoma to which our case belongs, we find 2 other cases—one of Chenantais,[1] described as a medullary alveolar neuroma, and Neumann's (already mentioned)—which, I think, may safely be included in Birsch-Hirschfeld's "endothelial sarcomata." (Endotheliomata are the tumors which are most likely to present an alveolar structure; thus it is to be found in 3 of the 5 cases reported—in Knapp's,[2] in Alt's,[3] and in Evetsky's.[4])

The extension of the tumor into the interior of the eye is a very rare occurrence, we only find it recorded in four cases: (1) in a case of Goldzieher's [5] of glioma of the optic nerve and retina, in which the optic-nerve tumor may very well have been secondary to the retinal; (2) a case of Knapp's [6] of sarcoma of the orbit, piercing the optic-nerve sheath and extending along the optic nerve into the eye, where it spreads in the nerve-fibre layer of the retina; (3) a case of Armaignac [7] of encephaloid of the retina and optic nerve (glioma), in which the retinal growth was most probably the primary one; and (4) a case of Sattler's [8] of a tubercular tumor of the optic nerve which extended into the eye along the nerve. Jacobson's case, usually quoted as a case of extension of an optic nerve-tumor into the interior of the eye, does not, as already stated, belong here, as in it there were scattered myxomata in the orbit with metastatic foci in the eye, and no optic-nerve tumor proper.

Clinical Features of the Case.—There is nothing of special interest about these. The symptoms present: exophthalmos in a directly forward direction, early loss of sight, painless course, optic neuritis, hypermetropia, increased tension, etc., being common to most optic-nerve tumors; generally there is more mobility preserved than in this case. It would have been interesting to note the ophthalmoscopic picture presented by the nerve head on its invasion by the neoplasm. It is a pity that during the later stages of the disease the opacity of the cornea prevented an ophthalmoscopic examination; an addition to Jacobson, Knapp, and Sattler's descriptions would have been instructive.

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[1] *Soc. Anat. de Nantes*, 1879.

[2] ARCH. OF OPHTH. AND OTOL., iv., 323. This case was described as a carcinoma, but a careful study of the case and drawings leads me to conclude that in all probability it was an endothelioma. At the time when the case was reported endothelioma had not yet been properly studied.

[3] *Arch. f. Augenheilk.*, vii., 1, 46.

[4] *Arch. f. Augenheilk.*, xii., 1, 16.

[5] *Arch. f. Ophth.*. xix., 3, 134.

[6] ARCH. OF OPHTH. AND OTOL., v., 132.

[7] *Journ. de méd. de Bordeaux*, 1878

[8] *Arch. f. Ophth.*, xxiv, 3, 127.

STATISTICAL CONSIDERATIONS.

I think it may be of interest to some if, before terminating, I call attention to a few points of general statistical import as regards the age of appearance and duration of these tumors, as well as to the results obtained by the different methods of operative interference as inferred from the appended tabular report.

Age.—Optic-nerve tumors have been found at all ages; from almost immediately after birth to sixty-seven years of age. In the sixty-seven cases where the age is mentioned we find:

Before 10 yrs.	30 cases.
Between 10 and 20 yrs.	16 "
" 20 " 40 "	14 "
" 40 " 60 "	5 "
After 60 yrs.	2 "

Youth is thus seen to be a marked predisposing factor; this is especially true for tumors of the sarcoma type. All the gliomata except one occurred in patients under 10; the one exception was a glio-sarcoma.

Duration.—The duration of the tumors before operative interference or before death, is mentioned in 50 cases. It fluctuates between 2 months and 20 years,—the average duration being 4.61 years. The course is apt to be slower with myxomata, endotheliomata, and fibromata. The following table will show the relative duration of each kind of tumor:

NATURE.	EXTREME DURATION.	AVERAGE.
Fibromata [1]	From 4 mos. to 20 yrs.	5.20 yrs.
Myxomata	" 2 yrs. to 14 "	7.10 "
Sarcomata [2]	" 3 mos. to 18 "	3.47 "
Gliomata	" 6 " to 2 "	1.50 "
Endotheliomata	" 3 " to 8 "	5.83 "
Carcinoma	" to 8 mos.	.66 yr.
Tubercle	" 2 " to 6 yrs.	3.08 yrs.
Tumors without denomination	" 2 yrs. to 8 "	4.40 "

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[1] We include in this group the myxo-fibromata.

[2] We include in this group the fibro-, myxo-, and endothelial sarcomata.

Operative Results.—Three principal operative procedures have been employed in dealing with these tumors; (*a*) exenteration of the orbit; (*b*) extirpation of the eyeball and tumor; (*c*) extirpation of the tumor with preservation of the eyeball. An operation is recorded in 68 cases. An exenteration was performed in 3 cases, all of which recovered, and in none of which there was any recurrence.

In 54 cases both eyeball and tumor were extirpated; mention of the subsequent course is made in only 25 cases; there was no recurrence in 10, the neoplasm recurred in 8 cases, and in 7 the operation was followed by death within a few days, generally with cerebral symptoms. This can in most cases be attributed to sepsis. In 4 of these cases metastatic growths or an extension into the brain were, however, found. It is safe to infer that there was no recurrence in the majority of the 29 cases where no mention is made of the subsequent course.

The eyeball was preserved in 11 cases.[1] Knapp is the surgeon who has performed most of these. There was no recurrence in 4 cases; one case was followed by death within a few days. The eyeball was subsequently lost or had to be enucleated in 4 cases, in one of which the tumor recurred, and in one of which the operation was followed within a few days by death. In this case there was an intracranial extension of the growth.

We find mention made of operations performed on recurring tumors in 4 cases: 2 recurred again, and in 2 the operation was followed by death within a few days. In one of these an intracranial extension was found at the autopsy.

Thus we find in 68 cases 11 relapses and 11 deaths. The relapses may, in view of the poor statistics yielded by the secondary operations, be considered to have terminated fatally. The recurrences occurred in the cases of: 1 fibroma[2]

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[1] Critchett, *Med. Times and Gazette*, 1832; Knapp, ARCH. OF OPHTH. AND OTOL., iv., 323; Knapp, *Ophth. Soc.*, Heidelberg, 1874; Knapp, ARCH. OF OPHTH. AND OTOL., v., 132; Grüning, ARCH. OF OPHTH. AND OTOL., v., 508; Schott, *Arch. f. Augenheilk.*, vii., 1–81; Alt, *Arch. f. Augenheilk.*, vii., 1, 46; Strawbridge, *Trans. Am. Ophth. Soc.*, 1878; Knapp, *Trans. Am. Ophth. Soc.*, 1879; Rohmer, *Soc. franç. d'opht.*, Mai 2–5, 1892, *Ann. d'Oc.*, cvii., 354; Lagrange, *Congrès franç. d'opht.*, April, 1892, *Ann. d'Oc.*, cvii., 377.

[2] A secondary operation performed on the recurring growth was followed within a few days by death.

(of 20 years' duration); 1 sarcoma; 1 fibroma-sarcoma (of 2 years' duration); 1 myxo-sarcoma; 1 endothelioma (of 8 years' duration); in 1 case of tubercular tumor,[1] 1 glioma,[2] 2 of unnamed tumors (one of 4 years' duration). The deaths occurred in: 2 sarcomata (one of 3½ years' duration); 1 fibro-sarcoma (of 2 years' duration); 2 myxo-sarcomata (of 3 and 5 years' duration); 1 endothelial sarcoma (of 18 years' duration); 2 gliomata (one of 2 years' duration); 1 glio-sarcoma (of 2 years' duration), and the 2 cases following secondary removals.

These statistics may be summarized as follows:

Operation.	Recurrence.	Death from Operation.	Recorded Non-Recurrence.
Exenteration of the orbit, 3 cases.			3 cases.
Extirpation of tumor and eyeball, 54 cases.	8 cases.	7 cases.	10 "
Extirpation of tumor, with preservation of eyeball, 11 cases.		1 case.	4 "
Subsequent loss of eyeball, 4 cases.	1 case.	1 "	
Secondary operation, 4 cases.	2 cases.	2 cases.	

CONCLUSIONS.

These statistics teach us that an operative interference is called for as soon as the diagnosis is made, as the tumor, if left to itself, will almost necessarily produce death, and the earlier the operation is performed the greater the chance of success. The comparatively large proportion of deaths immediately after the operation makes a strict adherence to the rules of aseptic and antiseptic surgery a necessity. The presence of metastatic growths within the cranium or of a direct intracranial extension seems to predispose to infection of the meninges; if such an extension can be inferred, all operative interference, with a radical cure in view, is contra-indicated.

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[1] This should not be considered as a real recurrence. The patient died after some time of a tubercular meningitis.

[2] In this case three operations were successively performed, the three being followed by a recurrence of the growth.

In view of the good statistical returns, an attempt at preserving the eyeball is justifiable and even recommendable, provided an ophthalmoscopic examination permits one to infer that there is no extension of the growth into the interior of the eye, and that the presence of the eyeball does not interfere with the complete excision of the tumor by limiting the field of operation.

Where there is any suspicion of an extension into the orbit an exenteration should be performed.

The results of operations on recurring growths are not encouraging, but I think that an exenteration of the orbit may be at times beneficial, provided there are no grounds to suspect an intracranial extension.

I take pleasure in expressing my thanks to Dr. E. Lopez for the pathological specimen which forms the basis of this paper; to Dr. C. M. Desverinne for the use of his laboratory; and to Drs. Santos Fernandez, Montalvo, and Lopez for the use of their libraries.

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
1	Blegny	Zodiacus medico-gallicus, p. 81 (quoted by Morgagni, see below).	Calcareous concretion in optic nerve.					
2	Morgagni	13th letter (quoted by Demarquay—Traité des tumeurs de l'orbite, Paris, 1860).	Cyst of optic nerve.					
3	Morgagni	13th letter.—Art. 6,(Demarquay)	Calcareous concretion in optic nerve.					
4	Scarpa	(Demarquay.)	Scirrho-cancerous (?) tumor of orbit originating in optic nerve					
5	Astley Cooper	Velpeau, Méd. opératoire, 2e edition. ii., 212.	Aneurism of central artery of the retina.					
6	Demours	Traité des maladies des yeux, i., 75.	Tubercular tumor of optic nerve.	36 yrs.	2 mos.			
7	Schaeffner	Recueil de thèses Allemandes. (Demarquay.)	Cancerous(?) degeneration of optic nerve.					
8	Paw	(Wardrop,—see below.)	Hydatid cyst of optic nerve.					
9	Heavyside	(Wardrop,—see below.)	Tumor of optic-nerve sheath.					In Mr. Heavyside's museum
10	Wardrop	Essays on the morbid anatomy of the eye. ii.,-1834.	Cancerous (?) degen. of opt. n.					
11	Manoir	(Demarquay.)	Cancerous (?) degen. of opt. n.					
12	Shayer	(Demarquay.)	Cancerous (?) degen. of opt. n.					
13	Eliason	(Demarquay.)	Cancerous (?) degen. of opt. n.					
14	Panizza	(Demarquay.)	Cancerous (?) degen. of opt. n.					
15	Middlemore	(Demarquay.)	Cancerous (?) degen. of opt. n.					
16	M. Roux	(Demarquay.)	Cancerous (?) degen. of opt. n.					
17	———	Schmidt's Jahrbuch i.,-352	Optic nerve tumor.		2 yrs.	Enucleation and extirpation of tumor.		
18	Lebert	Maladies cancereuses, p. 841.	Cancer (?) of optic nerve.					
19	Lebert	Ibid.	Cancer (?) of optic nerve.					
20	Lebert	Ibid.	Cancer (?) of optic nerue.					
21	Lebert	Ibid.	Cancer (?) of optic nerve.					
22	Lebert	Ibid.	Cancer (?) of optic nerve.					
23	Critchett	Med. Times and Gaz., 1832.	Fibrous tumor of optic nerve.		15 mos.	Extirp. of tum. with preserv. of eyeball.		
24	Schott	Controverse über die Nerven des Nabelstrangs, 1836, also Ed. Med. and Surg. Journ. xi.,274.	Fungus hæmatoides, connected with optic nerve.			Exenteration of orbit.	No recurrence 7 years later.	
25	Middlemore	Gaz. médicale, 1838.	Scirrhus (?) orig. in opt. n. sh.					
26	Gosselin	Bull. de la Soc. de Chirurg. viii., 112.	Several cases.					
27	Schmidler	Dict. des sc. méd., xxxv., 20.	Aneurism of central art. of ret.		Blind sev. yrs.			Princess of Baden.
28	Walter	Musée anat. (Demarquay.)	Calcareous plate in optic nerve.					

No.	Author	Publication	Nature of tumor	Age	Dur.	Operative procedure	Results	Remarks
29	WALTER	Bull. de Therap., 1844.-xxvii., 25.	Gelatiniform cancer (?) (myxoma (?)).	13 yrs.	4 yrs.	Extirp. of tum. and eyeball.		
30	MACKENZIE	Diseases of the Eye. 1844.	Fibrous tumor.		2 yrs.	Extirp. of tum. and eyeball.	No recurrence 20 months later.	
31	DIXON	Med. Times and Gaz. 1858. (Parisotti and Despagnet, see below.)	Gumma of optic nerve.					
32	BÖHM	Dublin Quart. Journ. of Med. Sc., Feb., 1847.	Fibrous tumor.	19 yrs.	13 yrs.			Death from pulmo. phthisis found at autopsy.
33	RITTERICH	Weitere Beiträge zur Vervolkommung der Augenheilkunst, 1861–57.	Sarcoma.	4 yrs.	3½ yrs.	Enucleation and extirpation of tumor.	Death with cerebr. sympts. 41 hrs. later.	Traumatism with forceps at birth. Tumor extended to chiasma.
34	SKOKALSKI	Ann. d'ocul., xlvi., 43.	Scirrho-cancerous tumor (?).	4 yrs.	4 yrs.	Enucleation and extirp. of tum.	Reccurence at end of 5 yrs, filling orbit.	Recurring tum. operated by other surgeon, purulent meningitis, death on 6th day, extension into intra cranial portion of nerve.
35	ROTHMUND (Jun)	Zeh. klin Monatsbl. 1863., 262.	Cystoid myxoma.	15 yrs.	13 yrs.	Enucleation and extirp. of tum.		
36	v. GRAEFE	Arch. f. Ophth., x., 1, 193.	Myxoma.	23 yrs.	2 yrs.	Enucleation and extirp. of tum.		
37	v. GRAEFE	Arch. f. Ophth., x., 1, 201.	Myxoma.	29 yrs.	7 yrs.	Enucleation and extirp. of tum.		
38	GALEZOWSKI	Thèse de Paris., 1865.	Fibroplastic tumors in optic n. and brain.					Found at an autopsy.
39	v. GRAEFE	Arch. f. Ophth., xii., 2–100.	Glioma.	6 yrs.	2 yrs.	Enucleation and extirp. of tum.	Death from purulent mening. (13th day.)	Exten. of tum. to incr. portion of both nerves. and to corp. str.
40	LIDDELL	N. Y. Med. Journ., 1866.	Neuroma (?).	20 yrs.	8 yrs.	Exenteratio orbitæ.	No recurrence after 14 months.	No microscopic examination.
41	SICHEL (fils).	Gaz. hebd., 1871.	Myxoma gelatinosum.	16 yrs.	2 yrs.	Enucleation and extirp. of tum.	No recurrence after 2 years.	
42	HORNER	Correspondenzbl. f. Schw. Aerzte, April 1, 1871.	Myxo-sarcoma of optic nerve.			Enucl. and extirp. of tumor.		
43	QUAGLINO	Ann. di Ottalm., i., 1, 27 and 3, 337, 1871.	Myxo-sarcoma.	24 yrs.	5 yrs.	Enucleation and extirp. of tum.	Death 6 months after opera. with cerebral symptoms.	Traumatism of 5 years before.
44	NARKIEWICZ-JODKO	Nagel's Jahresbericht., 1872, 363.	Myxoma.	11 yrs.		Enucleation and extirp. of tum.		

45	ARON HEYMAN	"De neuromate nervi optici."—Berlin, 1872.	True neuroma (?).	19 yrs.				
46	KRAHM	Zeh. klin Monatsbl., 1872, 103.	Carcinoma (metastatic).	30 yrs.	8 mos.			Found at an autopsy. Both nerves presented tum. Carcin. tum. of both ovaries. Found at an autopsy.
47	GOLDZIEHER	Arch. f. Ophth., xix., 3-125.	Fibroma myxomat.	10 yrs.		Enucleation and extirp. of tum.	No recurrence 1 year later.	
48	GOLDZIEHER	Arch. f. Ophth., xix., 3-134.	Glioma with myxomat. deg.	4½ yrs.	6 mos.	Enucleation and extirp. of tum.		*Extension into retina.*
49	GOLDZIEHER	Arch. f. Ophth., xix., 3-139.	Myxo-sarcoma.	4 yrs.		Enucleation and extirp. of tum.	Relapse in orbit 1 year later; 8 years later child blind of other eye and paralyzed.	
50	STEFFAN	Augenheilanstalt, Francfort, 1873 33.	Fibro-sarcoma.	30 yrs.		Enucleation and extirp. of tum.	No recurrence 15 months later.	
51	PERLS & LOCH	Arch. f. Ophth., xix., 2, 287, 1873	True neuroma (?).	9 yrs.	2 yrs.	Enucleation and extirp. of tum.	No recurrence 5 months later.	Traumatism 2 years before.
52	KNAPP	Arch. f. Ophth. and Otol., iv., 323. 1874.	Carcinoma (?) of outer sheath of optic nerve (endothelioma ?).		3½ yrs.	Extirpation of tum. with preservation of the eyeball.	No relapse 8 months later. (Patient living and healthy, eye shrunken, painless. No relapse 1894.—H. K.)	S preserved. Pt. under observation for 3 yrs. before oper.
53	KNAPP	Ophth. Soc. Heidelb, 1874. (Ann. d'Ocul., 1875.)	Fibro-sarcoma.	40 yrs.	3 yrs.	Extirpation of tum. with preservation of the eyeball.		
54	DUSAUSAY	Bull. de la Soc. Anat. x., 1875.	Angiolithic sarcoma (Endothelial sarcoma ?).	50 yrs.	18 yrs.	Enucl. and extirp. of tumor.	Profuse suppur. Death w. cereb. sympts. (8 days).	
55	CHRISTENSEN	Nagel's Jahresb., 1875, 386.	Tumor of optic nerve.	21 yrs.		Euncl. and extirp. of tumor.		Tumor occupied intevag. space
56	KNAPP	Arch. of Ophth. and Otol. v., 132.	Large orb. sarcoma. Optic nerve affected secondarily.	38 yrs.		Removal of tumor w. preserv. of eyeball.	Death on 12th day fr. acute nephr.	*Extension into the interior of the eye along opt. n.* Metastases in liver and testicle. Croupous nephr.
57	GRÜNING	Arch. of Ophth. and Otol., v., 508.	Myxoma.	34 yrs.	14 yrs.	Extirp. of tumor w. preserv. of eyeball.	11 yrs. later pt. alive and eyeball still preserved (Knapp).	History of fall.
58	NEUMANN	Arch. of Heilk., xiii., 310 (Knapp's report in Trans. Internat Congress, Phila., Sept., 1876).	Sarcoma (Psamoma, Endothelial sarcoma ?)	20 yrs.	3 yrs.	Extirp. of tumor and eyeball.		Structure partly alveolar, partly compact.
59	REICH	Arch. of Ophth., xxii., 1, 103.	Endothelioma.	12 yrs.		Enuc. and extirp of tumor.		
60	SCHOTT	Arch. of Ophth. and Otol., vi., 276.	Endothelioma (both nerves).	55 yrs.				Found at an autopsy.
61	HOLMES	Arch. of Ophth. and Otol, vi., 299.	Myxo-fibroma.	10 yrs.	4 mos.	Removal of eyeball and tumor.		
62	ALT	Arch. f. Augenh., vii., 1, 46.	Endothelioma.	31 yrs.	6 yrs.	Extirp. of tum. w. preserv. of eyeball.	Enucleation subseq. necessary.	

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
63	SCHOTT	Arch. f. Augenh., vii., 1, 81.	Glio-sarcoma.	3½ yrs.	2 yrs.	Removal of tum. w. preserv. of eyeball.	Enucl. subseq. necessary. Death on 7th day fr. pur. meningitis.	Chiasma and other nerve invaded.
64	BRAYLEY	Ophth. Hosp. Reps., ix., 11, 123.	Fibróma.	67 yrs.	2 yrs.	Enucl. and extirp. of tumor.		History of blow.
65	ARMAIGNAC	Journ. de méd. de Bordeaux, 1878.	Encephaloid of ret. and opt. n. (glioma ?).	26 mos.		Enucl. and extirp. of tumor.	3 removals and 3 recurrences.	*Involvement of the retina* seems primary.
66	FORSTER	Arch f. Ophth., xxiv., 2, 103.	Fibro-nuclear tumor.	15 yrs.	3 yrs.	Exenteratio orbitæ.	No recurrence 1½ yrs. later.	
67	PUFAHL	Breiträge z. prakt. Augenheilk., v. Hirschberg, 1878, iii., 63.	Sarcoma.	14 yrs.		Enucl. and extirp. of tumor.		
68	STRAWBRIDGE	Trans. Am. Ophth. Soc., 1878, 383.	Tumor of optic nerve (glioma ?).	31 yrs.		Extirp. of tum. w. preserv. of eyeball.	Subseq. loss of globe. S. of other eyes subseq. lost w. cerebr. sympts.	
69	SATTLER	Arch. f. Ophth., xxiv., 3, 127	Tubercular tumor of optic nerve.	5 yrs.		Enucl. and extirp. of tumor.	Death 5 months later from tubercular meningitis.	*Extension into retina.*
70	LEBER	Arch. f. Ophth., xxv., 189.	Myxo-sarcoma.	6 yrs.	18 mos.	Enucl. and extirp. of tumor.		
71	LEBER	Arch. f. Ophth., xxv., 195.	Myxo-sarcoma.	4½ yrs.	3 yrs.	Enucl. and extirp. of tumor.	Death 9 days later w. mening. sympts.	Extirp. incomplete due to intra cranial extension. Invasion of chiasma, foci in other nerve.
72	LAQUEUR	Arch. f. Ophth., xxv., 241.	Myxo-sarcoma.	14 yrs.	1½ yrs.	Enucl. and extirp. of tumor.	No recurrence 15 mos. later.	
73	HIGGENS	Brit. Med. Journ., Oct. 18, 1879, 616.	Fibroma.	5 yrs.	2 yrs.	Enucl. and extirp. of tumor.		
74	KNAPP	Trans. Am. Ophth., Soc., 1879, 557.	Myxoma.	2 yrs.	2 yrs.	Extirp. of tumor. w. preserv. of globe.	Eyeball subseq. lost thro' phthisis 9 mos. later S. of other eye lost by desc. neuritis. (Propagation or symp. ophth.?)	
75	CHENANTAIS	Soc. anat. de Nantes, 1879.	Med. alv. neuroma (sarcoma ?).	18 yrs.	3 mos.	Enucl. and extirp. of tumor.		
76	EVETSKY	Arch. f. Augenh., xii., 1, 16.	Endothelioma.		8 yrs.	Enucl. and extirp. of tumor.	Recurrence 8 mos. later, filling whole orbit.	
77	RAMPOLDI	Congrès internat. d' opht., Milan, 1881.	Glio-sarcoma.			Enucl. and extirp. of tumor. (Quaglino.)		
78	PONCET (Cluny)	Arch. d' opht., 1881, 616.	Fascicular myxoma.	16 yrs.	13 yrs.	Enucl. and extirp. of tumor. (de Wecker).		
79	TEILLAIS	Journ. de méd. de l' Ouest-Nantes, 1881, xv., 74.	Glioma (?) (Myxo-sarcoma ?).	62 yrs.	7 yrs.	Enucl. and extirp. of tumor.	No recurrence 3 yrs. later.	
80	LEBER AND VOSSIUS	Arch. f. Ophth., xxviii., 3, 33, and 283.	Myxo-sarcoma.	2½ yrs.	1½ yrs.	Enucl. and extirp. of tumor.	No recurrence 3 yrs. later.	
81	LEBER AND VOSSIUS	Ibid.	Myxo-sarcoma.	8 yrs.	1 yr.	Enucl. and extirp. of tumor.	No recurrence 9 mos. later.	
82	V. RECKLINGHAUSEN	Ueber die multiple Fibrome der Haut, u. s. w. Festschrift, Berlin, 1882.	Angiolithic fibroma.					Tumor at Strasburg Institute.

83	Lawson	Royal London Ophth. Hosp. Reps., 1882, 296.	Sarcoma of optic nerve sheath.	65 yrs.		Enucl. and extirp. of tumor.	Recurrence 1 mo. later. Death 3 mos. later.	Metastases in bronch. and mesent. glds., pancreas, duodenum, spleen, and liver. Sec. deg. of r. kidney. Ant. ⅔ of brain surface covered w. coag. lymph. foci of softening.
84	Huc	Thèse de Paris, 1882.	Fibro-sarcoma.	7 yrs.	2 yrs.	Enucl. and extirp. of tumor.	Death on 9th day. Cerebr. sympts.	History of fall. Chiasma invaded.
85	Hulke	Royal London Ophth. Hosp. Reps., 1882.	False neuroma (Sarcoma?)	19 yrs.	6 yrs.	Enucl. and extirp. of tumor.		
86	Veron	Rec. d'opht., 1883, 32.	Myxo-fibroma.	18 yrs.	3 yrs.	Enucl. and extirp. of tumor.		
87	Peabody	N. Y. Med. Record, Feb., 1883.	Sarcoma.					Found at an autopsy. D. fr. mening.
88	Roux	Gaz. des hôp., Aout 6, 1884.	Myxomat. tumor.	13 yrs.	4 yrs.			Pathological specimen.
89	Johnson and Prudden	Arch. of Ophth., xiv., 2, 151.	Myxo-sarcoma.	12 yrs.	6 yrs.	Extirp. of tumor and eyeball.		
90	Parisotti & Despagnet	Rec. d'opht., 1884, 725.	Fibroma.	31 yrs.	20 yrs.	Enucl. and extirp. of ant. port. of tumor.	4 yrs. later remainder incr. in size: exenteratio orbitæ. Death 2 days later.	
91	Kunachowich	Med. Obezz., Moscow, 1885, xxiv., 293.	Myxoma.	18 yrs.	10 yrs.	Enucl. and extirp. of tumor.		
92	Vossius	Berlin. klin. Wochenschr., Mar. 30, 1885.	Myxo-sarcoma.	20 yrs.	1 yr.	Enucl. and extirp. of tumor.		
93	Brayley	Sem. Méd., Nov. 24, 1886.	Sarcoma.	42 yrs.		Enucl. and extirp. of tumor.		
94	Panas	Jocqs, Thèse de Paris, 1887.	Tumor of optic n. (Giant-celled tum. tubercular?).	59 yrs.	6 yrs.	Enucl. and extirp. of tumor.	No recurrence.	
95	Tillaux	Leçon clin. reç. par. M. Guillet, Gaz. des hôp., Fev. 17, 1887.	Primary tumor of optic nerve (Sarcoma).	40 yrs.	18 mos.	Enucl. and extirp. of tumor.		Jocqs, in his thesis, believes that if left, myxom. deg. would have set in.
96	Frothingham	Am. Med. Assoc. (Arch. d' opht., 1887, 480).	Sarcoma.					
97	Frothingham	Journ. Am. Med. Assoc., Dec. 10, 1887 (Arch. d' opht, 1888, 96).	Tumor of optic nerve.					
98	Frothingham	Ibid.	Tumor of optic n.					
99	Lawson	Roy. London Ophth. Hosp. Repts., xii., Jan., 1888.	Myxoma.					
100	Sutphen	Trans. Am. Ophth. Soc., 1889.	Sarcoma.	10 yrs.				

No.	Author	Publication	Nature of tumor	Age	Durat.	Operative procedure	Results	Remarks
101	CIRINCIONE	Giorn. di neuropatologia, vii., 2.	Tubercular tumor.					
102	AYRES	Am. Journal of Ophth., March, 1890.	Myxo-sarcoma.	12 yrs.				
103	AYRES	Ibid.	Sarcoma.	22 yrs.				
104	KESSLER	Weekbl. der Nederland Tijdsch. v. Geneeskunde, ii., 781, 1891.	Intravaginal sarcoma of optic nerve (fibrosarc w. myx. deg.).	5 yrs.	18 mos.	Enucl. and extirp. of tumor.		
105	GARNIER	Zeh. klin. Monatsb. f. Augenh., xxix., 208.	Myxo-sarcoma.					
106	DITTRICH	Prager Vierteljahrschr., xii. (quoted by Elschnig).	Multiple cancerous foci in opt. nerve as well as in brain and other cerebr. nerves.					
107	ELSCHNIG	Arch. of Ophth., xx., 3, 370.	Metast. carcinoma of intracr. port of opt. n. extending into orbitæ.					Carcinoma renis
108	KEYSER	Ann. of Ophth. and Otol., 1892	Glioma.	8 yrs.		Enucl. and extirp. of tumor.	Death on 5th day w. cereb. sympts.	
109	ROHMER	Soc. franç. d'opht., Mai, 1892 (Ann. d'Oc., cvii., 354).	Telangiectatic tumor of optic nerve.	3½ yrs.		Extirp. of tumor w. preserv. of the eyeball.	No recurrence 18 mos. later.	
110	LAGRANGE	Congrès franç de chirurg, April, 1892 (Ann. d'Oc., cvii., 377).	Myxo-sarcoma.	13 yrs.	3 mos.	Extirpat. of tum. w. preserv. of eyeball.	No recurrence 6 mos. later.	
111	BOURGEOIS	Soc. d'opht. de Paris, Sept. 10, 1893.	Myxo-sarcoma of opt. n. and orbit.			Enucl. and extirp. of tumor.		
112	BRAUNSCHWEIG	Arch. f. Ophth., xxxix., 1 (Extr. of Arch of Ophth., xxiii., 3, 354).	Tumor of optic nerve.					
113	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
114	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
115	BRAUNSCHWEIG	Ibid.	Tumor of optic nerve.					
116	ROCKLIFFE	Trans. Ophth. Soc. of U. Kingd., xiii., 101.	Tumor of optic nerve.			Enucl. and extirp. of tumor.		
117	FINLAY	*This paper.*	Alveolar fibro-sarcoma.	55 yrs.	2 yrs.	Enucl. and extirp. of tumor. (Lopez).	Death 1 yr. later w. cereb. sympts. For some time pt. had been idiotic and blind of other eye which showed total opt. n. atrophy.	*Extension into interior of eye.* Structure partly compact, partly alveolar, and partly myxomatous.

NOTE.—In collecting these cases I have frequently been unable to cousult the original papers and have had to be contented with extracts; as we are, here in Cuba, unfortunately very scantily supplied with ophthalmological literature, I have mostly utilized those of the *Archives of Ophthalmology* and those of the *Annales d'Oculistique*. Dr. Jocqs's valuable thesis has also been of great help to me in this particular. In view of this I trust I will be pardoned any inaccuracy that may have crept in.—C. E. F.

TRAUMATIC PARALYSIS OF THE ABDUCENS NERVE.

By C. ZIMMERMANN, M.D., Milwaukee, Wis.

SINCE the publication of the articles of Purtscher and Friedenwald in these ARCHIVES (xxiii., No. 4), in which the authors collected the reported cases of traumatic paralysis of the abducens nerve (fifty-seven, with their own), two further cases of unilateral traumatic basal paralysis of the abducens have come to my notice.

CASE 1 is described by Professor A. Eulenburg (*Neurolog. Centralblatt*, 1894, No. 16, and Litteratur-Beilage, der *Deutsch. Medic. Woch.*, 1894, No. 14, p. 102):

A man, aged thirty-two, was stabbed with a knife in the right temple. A copious hemorrhage followed immediately and he became unconscious. After a few weeks complete paralysis of the right abducens nerve was ascertained, and a slight degree of right-sided ptosis, which disappeared later on; inconsiderable impairment of vision, headache, and vertigo. Eulenburg assumes that the blade must have injured the sixth nerve, where it is exposed to an isolated lesion, apart from the other (motor and sensitive) ocular nerves—*i.e.*, before its entrance into the superior orbital fissure, and where, in its course toward the basilar clivus, laterally and behind the dorsum sellae, it enters the cavernous sinus, and runs on the lateral side of the carotis interna. The ptosis may have been due to a slight lesion of the third nerve on its passage in the superior wall of the sinus.

CASE 2 came under my own observation:

April 26, 1894—Thosten Thomsen, a Norwegian ship-carpenter, aged forty-seven, was struck by a man with a capstan bar on the left side of his head, a few *mm* above a point midway between the external auditory meatus and the occipital protuberance. He went down-stairs to the cabin, but then he fell unconscious, and did not recover until the next day, while he was lying in the hospital. Right after the blow blood came out of the ear. Later he was told by the nurse that in the evening after the accident he was brought to the Emergency Hospital, and that he vomited blood. The next day he recovered consciousness, but felt sick, and suffered from diplopia and vertigo; could not raise his head; or, when raised, could not hold it up. April 27th he was transferred to the Marine Hospital, where he lay in bed for ten days. Dr. J. M. Allen sent him to me for an examination of his eyes (May 12th). I found complete paralysis of left abducens nerve. Homonymous diplopia, even in right field of fixation, from increased contraction of internal rectus. Pupillary reaction normal. V R = $\frac{15}{15}$—V L = $\frac{15}{20}$ with—0.75 D = $\frac{15}{10}$. V F normal. No ophthalmoscopic changes. The other cranial nerves, especially the facial and acoustic, not affected.

Left ear: Ordinary voice at eight feet; watch not at all. T. F. better opposite the ear than at mastoid process. The posterior wall of meatus and the posterior half of *Mt* are covered by dried blood. This feels hard to the touch with the probe, which is painful. After removal of the blood on May 25th no fracture of the osseous canal can be detected, but the *Mt* shows a vertical line between umbo and posterior margin, apparently the scar of a rupture, through which the blood had escaped. Tinnitus, headache, and vertigo. He cannot balance himself well, when he walks, feels "topheavy," as he expresses it, even when the left eye is tied up. June 1st he had chills and had to stay in bed for a day. While I observed him until July 2d, and as long as he stayed in the hospital until August 17th, his condition did not change, and is still the same, as a letter of February 3d from Dr. F. Gudmundsen, of Detroit Harbor, Wis., where the patient now lives, informed me—*i.e.*, the paralysis is permanent.

In this case the primary paralysis of abducens was undoubtedly of basal origin, due to a transverse fracture of the apex of the petrous bone, tearing or lacerating the nerve at this point in its course. The hemorrhages from ear and mouth directly after the blow corroborate this diagnosis. That the fracture must have been a transverse, not a longitudinal one, becomes clear from the facial and acoustic nerves not being involved, and from the action of the force on the same side of the head in the line of the sagittal suture. The impairment of hearing was not due to an affection of the acoustic nerve itself, but rather to a lesion of the labyrinth and the middle ear, into which an effusion of blood had occurred.

A CASE OF ANOMALOUS DIPLOPIA, DIVERGENT STRABISMUS WITH HOMONOMOUS DIPLOPIA; TWO "FIXATION-SPOTS" IN THE SAME EYE.

By N. DARRELL HARVEY, M.D., Providence, R. I.

SINCE hearing the report of Dr. Thomas of Philadelphia on "Three Cases of Strabismus with Anomalous Diplopia," made at the last meeting of the American Ophthalmological Society in Washington, I have had an opportunity of examining a case of the same class. Such cases are exceedingly rare, no mention of their occurrence being made in most of the leading text-books, so that they become of interest both upon this account, as also from the standpoint of treatment.

Besides these cases of Dr. Thomas, others have been described by Swanzy, Berry, Theobald, and Agnew.

The case I am about to report, although possessing many points of interest in common with those already reported, I think may yet present a few additional ones.

All the cases of Dr. Thomas were submitted to operation to rectify the deformity, and in none was orthophoria obtained. The present case has had no operation. The deformity has been so far removed, and a parallelism of the eyes apparently obtained by non-operative measures.

Although muscular equilibrium for all distances is not yet permanently established, orthophoria for near has been attained, and a similar condition for twenty feet, I think, will soon be found.

The gentlemen above referred to have suggested the presence of an additional "fixation-spot" in the deviating eye as the only solution of the problem of anomalous diplopia, and I feel that the facts of the following case add to the support of this theory.

The history of the case is this:

Miss B. L., seventeen, came May 5, 1894, complaining of pain and occasional swelling about the right eye. She stated the right eye had always squinted, but the deviation was more marked at times. She never remembers having had "double vision." She does not mind the deformity much, but wants relief from pain.

S. P.—General health is good; divergent strabismus of the right eye; her family physician can find no cause, unless it be her eyes, to account for all her trouble.

V O D $= \frac{5}{7.5}$ accepts + 0.50.0° (centrally). V O S $= \frac{5}{7.5}$ accepts + 0.25.90°. Ophthalmometer O D 0.25.0 — 90 +; O S 10 — 90 +. Accom. good. Fundus normal. With both eyes open and right one in divergence, patient has single vision far and near. On covering the left eye, the right one fixes centrally, but immediately returns to its former condition of divergence as soon as the left one is free. Diplopia cannot be induced by any of the usual methods.

There is not binocular vision for near, as shown by the stereoscopic test, but whether it exists for distance cannot be certainly stated.

May 24, 1894.—Under homatropine: V O D $= \frac{5}{6}$ — w + 1.0° (centrally). V O S $= \frac{5}{6}$ — w + 0.25.90°. Ordered Gl. + 1.0° = O D + 0.25.90° O S for constant use, and avoid close use of eyes as much as possible.

June 4, 1894.—Headache and pain about eye greatly relieved; still some pain remaining. Has Sp. accom. = 0.50. V $= \frac{5}{4}$ a o. To continue to wear glasses and exercise with pr. 12° base in, and endeavor to see "candle flame" at twenty feet double.

June 11, 1894.—Can now see "double" with vertical prism test and red glass. Eyes seem much straighter, and at times when the patient makes an effort there seems to be no real divergence. Under vertical diplopia test, the right eye being in divergence, she now has "homonymous diplopia," taking prism 3° b. out to produce horizontal equilibrium for distance, and pr. 6° b. out for near. With pr. 20° b. in, which corrects all movement of recovery of the right eye under the cover test she has the same homonomous diplopia. Advised to try "finger exercise" for the interni, for five minutes three times a day.

July 31, 1894.—Headaches very much less; formerly daily, now only ten in five weeks. Same muscular condition. To continue exercise as before.

October 10, 1894.—Eye has remained very straight, but of late turns, and more especially when she is bodily fatigued.

S. P.—No divergence now for far or near under the "cover test." With vertical diplopia test and red glass the right eye immediately diverges slightly, to a less extent than before, but a wider homonymous diplopia occurs for distance requiring now pr. 8° b. out to correct. She now has horizontal equilibrium for near.

Dynamical condition : Dist., ab. = 20°, ad. = 36°.
Convergence to 2-inch : Near, ab. = 20°, ad. = 26°.

She states that the eye feels better with pr. b. out, so she was ordered Gl. + 1.0° pr. 4° b. out. for the right eye,—same Gl. for the left.

November 3, 1894.—Prism has given more relief. Advised to continue as she is now doing.

November 16, 1894.—Adduction stronger,—feels well enough to begin work in dry-goods store. Advised to wear the glasses constantly, and use the "finger exercise" every day and report again in one month or three weeks.

December 7, 1894.—Has felt so well that she has not used the exercise much. Eye keeps straight most of the time. Abduction and adduction about the same as before. Under vertical diplopia test and red glass, eye diverges but slightly, now needing pr. 12° b. out to correct diplopia. With the same test and pr. 12° b. in, which now corrects all movements of recovery on the part of the right eye under the "cover test," after some few attempts both eyes continue to fix centrally, and the patient obtains horizontal equilibrium of her muscles for distance, getting the normal projection.

Now to sum up: the principal points of interest about this case are:

1. The presence of two "fixation-spots" in the right eye. The one, a false fixation-spot, being an eccentric hypersensitive portion of the retina to the temporal side, used in preference to the central spot entirely at first, but, since treat-

ment was begun, only under conditions which disturb the newly acquired binocular vision. The other, the true macula lutea, at first disregarded, except when the right eye was used alone, but, later, after parallelism of the eyes was acquired, concerned in the production of binocular vision.

2. The relief from pain, the correction of the deformity, and the acquisition of binocular vision by the correction of the refractive error, and the systematic exercise of the power of adduction without any operative interference. Inasmuch as the presence of a false fixation-spot is admitted, the question arises as to what extent it was concerned in binocular vision. From the above facts, while the eye was in divergence, such a condition of single vision, it would seem, did not exist. The image perceived by the right eye was mentally suppressed when both eyes were open, and only appreciated when the eye was made to fix centrally. But although not actively engaged in the production of binocular vision, this spot, nevertheless, did have a closer relationship to the macula of the other eye than did the central point, acting as the standpoint from which projection was estimated.

Under the stimulation which the tendency for binocular vision received from the mechanical exercise of the weak adductive power as well as the increased action of the innervation centres, the deviating eye became straight, and this false fixation-spot has given place to the true macula lutea, and is now regarded only under conditions which disturb binocular vision, and then only at times. As this function becomes more and more firmly established, the presence of the false spot is more and more disregarded, until finally we shall see the central spot predominant and normal projection maintained under all conditions.

Another singular point about the case is that the patient with weak adduction still obtains relief from a prism base out. This, I think, must be accounted for on the ground that there is a spasmodic esophoria, and the relief obtained from the prism base out is analogous to that seen from wearing a myopic cylinder, where there is a spasmodic myopic astigmatism due to weak accommodative effort, the true refraction being one of hyperopic astigmatism.

May 5, 1895.—As a further addition to the history of this case, I would add the following: The patient, partly from the fact that she has been feeling so well, as also forgetfulness, has neglected to use the exercise. Still, the eyes continue straight—pain and discomfort all gone. Orthophoria near and for short intervals for distance under the vertical diplopia test. Ordered to persist in her exercise.

CONGENITAL, MONOLATERAL FISTULA OF THE LACHRYMAL SAC, SITUATED ABOVE AND PARALLEL WITH THE INFERIOR CANALICULUS.

By JOHN DUNN, M.D., Richmond, Va.

Mr. L., aged 20, exhibits the following anomalous congenital opening into the right lachrymal sac. The inferior punctum is normal in size and appearance, it opens into the canaliculus, which in turn allows the passage of a probe into the lachrymal sac of this side. Directly internal to the punctum is a narrow slit, about 1½ *mm* long, through which liquid, if injected, passes into the lachrymal sac and thence into the nose. This secondary canal is directly above and parallel to the inferior canaliculus, and opens by its own mouth into the sac.

There is no communication between this secondary canal and the canaliculus. Two needle probes can be passed at the same time, one through each canal, into the sac. No pathological discharge can be forced from either canal. Mr. L. says that to his knowledge he has never had any inflammation of this eye. He is, however, from time to time annoyed with the accumulation of tears in the corner of this eye. What causes this accumulation of tears is difficult to say. It may be that the internal end of this secondary canaliculus, as well as its walls, are unprovided with any elastic tissue, and thus offer no resistance to the regurgitation of tears from the sac during the contractile efforts of the sac walls, or it may be that the presence of this canaliculus has prevented the proper development of the structures lying above the walls of the inferior canaliculus.

In the case of Mr. L. there is no scar tissue about the secondary canaliculus nor any evidence that this anomalous condition is the result of inflammatory changes. Nor is there any reason for believing that intranasal conditions cause the epiphora.

(*Cf.* "Congenital, etc., Fistulæ of the Lachrymal Sacs., by C. A. Wood, Archives of Ophthal., vol. xxiii., Nos. 1 and 2.)

A SECOND ATTACK OF PAPILLITIS OCCURRING IN A CASE OF POST-NEURITIC ATROPHY OF THE OPTIC NERVES.[1]

By G. E. de SCHWEINITZ, M.D., and A. G. THOMSON, M.D., of Philadelphia.

THE following case occurred in the practice of Dr. Wharton Sinkler, in the Orthopedic Hospital and Infirmary for Nervous Diseases, and we are indebted to him for frequent opportunities of examining the patient.

H. M. presented himself in the Out-Patient Department of the hospital on February 9, 1891, complaining of a series of epileptic attacks. His mother had died of some nervous disease; one brother was insane; his own mental state was good, but he had suffered much headache. There was no history of traumatism or syphilis, and no evidence of disease other than the nervous disorder.

The first attack occurred in December, 1889, the second nine months later, the third the next day, the fourth three months later, the fifth four months later, the sixth three weeks before presenting himself for treatment and the seventh the day before he came to the dispensary. These seizures manifested themselves soon after breakfast, the convulsive movements involving first the left arm, then the leg, face, and finally, the whole body.

The ocular examination at that time revealed the following points: Pupils normal; optic discs of good color; slight tortuosity of the retinal veins; very full lymph sheaths. Vision (direct and indirect) was normal. Macular region + 1 D.

On May 28, 1892, his eyes were again carefully examined. The discs were now slightly hyperæmic, their upper and lower edges veiled, the veins unusually full, and the lymph sheaths markedly infiltrated. Vision, accommodation, and pupillary reflexes were normal; there was slight esophoria, but no paralysis of any ocular muscle.

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[1] Paper read before the Section of Ophthalmology of the College of Physicians in Philadelphia, February 19, 1895.

After remaining under treatment for some months, he sought advice at the Hospital of the University of Pennsylvania, and while there was examined (March 4, 1893) in the eye dispensary by Dr. Wallace, who found double optic neuritis.

The next ocular examination, March 15, 1893, was made at the Orthopedic Hospital, and the following note was recorded: "Well-marked double optic neuritis; both discs swollen + 3 D; many fresh hemorrhages on and near the swollen nerve-heads; form and red fields normal. Vision, right, $\frac{20}{15}$; left, $\frac{20}{20}$."

The patient was admitted to the hospital in the hope of obtaining relief from his violent headache, which was persistent, as well as from the epileptic attacks, which had continued.

One month later, his general condition still being very good, the swelling of the right disc had increased to + 4 D, and that of the left to + 6 D. The eyes wandered in under cover, but the muscles were not paretic, neither was there diplopia; vision and field of vision continued normal. The patient was trephined, April 13th, by Dr. W. W. Keen over the right motor centres, a portion of the bone being removed. It was believed that the tumor was localized, but that it was not removable.

June 12, 1893. The patient has had less headache since the operation than during the past year. Eye examination on that day as follows: O D vision $\frac{20}{40}$, O S $\frac{20}{200}$; apex of the swelling in O D + 4 D subsiding on the temporal side; apex in O S + 6 D. In each macula degenerative changes, closely resembling those seen in renal retinitis, have appeared. Urine negative.

August 14, 1892. The patient had spasm on awakening from sleep. The head was jerked backward, the left arm convulsed; unconsciousness followed. This has been the first return of his convulsive seizures since the operation.

August 28, 1893. The apex of the swelling of the right disc was + 1 D, of the left + 4 D; margins much clearer than at previous examination; the exudation in the macular region was more marked and now extended from the macula to the disc.

October 30, 1893. The centre of the right disc was + 1 D, its outlines were clearly defined, with marked pallor on the temporal side. The swelling of the left disc was + 2 D, its outlines were appearing; the splotch of macular exudation was becoming atrophic and decreasing in size; the fundus on both sides, with the exceptions noted, was healthy. Vision, O D $\frac{20}{20}$, O S $\frac{2}{200}$. During all this time the patient's general condition continued to improve and he had returned to his usual work.

December 7, 1893. The vision of the right eye was normal, of the left $\frac{3}{200}$. The ophthalmoscopic appearances of typica post-neuritic atrophy were present, the atrophic pallor being chiefly noticeable in the temporal half of the right disc, while it was general in the left. In the macular region of each there was a patch of retino-choroiditis becoming atrophic upon the left side. The visual fields were normal in extent and the color perception perfect.

May 21, 1894.—The same conditions were present as at the last examination, the improved circulation in the nerve-heads being notable. Vision of the right eye $\frac{20}{20}$, of the left $\frac{20}{200}$.

During July the patient had one or two slight attacks, confined to the face, and complained of "a heavy feeling" in the arm.

Sept. 17th.—An attack occurred on the 8th of this month affecting the face and hands; since, there have been headache and pain over the right brow and behind the ear.

Early in November the patient was seized with violent pain in the head, associated with vomiting, and he noticed at once that vision in both eyes commenced to fail, the failure being somewhat gradual in its progress. There was weakness of the legs—not paralysis—and a sensation of general debility, confining him to bed until January 21, 1895, when he presented himself in the hospital, and the following note was made in regard to his eyes:

Movements of the eyes normal in extent in all directions, although when attempting to look directly forwards there is apparent divergence of the visual axes; there is no nystagmus; the pupils are dilated ad maximum, and fail to react to light or to attempts at convergence; there is absolute blindness. In the right eye the media are clear, the disc is swollen + 6 D (macula + 1 D), the swelling being of grayish color, and most marked above and below; the arteries are smaller than normal, the veins dark and somewhat tortuous, and carry white lines along their margins. In the left eye the disc is swollen + 6 D, the mound-shaped elevation being even more gray than upon the opposite side. The small patch of atrophic retino-choroiditis in the macula is still visible. The other portions of the retina appear normal.

In brief, then, we have the following conditions in this somewhat remarkable case, which has been observed for a period of four years: Focal epilepsy, creating the suspicion of intracranial tumor. At first there are no abnormal ocular symptoms except passive hyperæmia of the retinal circulation and infiltration of the lymph sheaths. One year later, the epilepsy continuing, there is distinct congestion of the optic disc, which, in a few months, develops into optic neuritis, and on the patient's return to the Orthopedic Hospital, two years after the primary examination, is evident as a well-marked double papillitis of the type ordinarily called "choked disc," without material disturbance in vision or the field of vision, and unassociated with abnormalities in the external ocular muscles or the pupillary reflexes.

From that time on, with the exception of decided increase in the papillitis and depreciation of vision in the left eye, the ocular conditions remained unchanged until the trephining, April 13, 1893.

Between three and four months subsequent to the operation, the ophthalmoscopic appearances of subsiding neuritis begin, and by December 7, 1893, or eight months after the operation, there is fully developed post-neuritic atrophy, with preservation of good vision in the right eye, and very moderate (one tenth of normal) vision in the left.

Nearly one year after this date the patient reappears with the return of headache, vomiting, and other symptoms of intracranial disease, from which he had been previously free, totally blind, and revealing to the ophthalmoscope well-marked double optic neuritis, the apex of the swelling being as high (namely, 6 D) as it had ever been in the primary attack.

Second attacks of optic neuritis in completely atrophied nerve-heads are exceedingly uncommon. Dr. Gowers,[1] commenting on cases of this character, refers to a boy, aged twelve, under the care of Dr. Hughlings Jackson, with double optic atrophy and absolute blindness from intracranial disease some years previously, in whom distinct double papillitis occurred in the atrophied discs, associated with symptoms of cerebral tumor.

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[1] *Medical Ophthalmoscopy*, 3d ed., p. 56.

Even when the atrophy is partial, or absent, according to the same authority, and this certainly is our experience, two attacks of neuritis are rare. Dr. Gowers describes a patient who suffered from cerebral tubercle and died from an attack of tubercular meningitis. The neuritis caused by the former lesion disappeared, leaving partial atrophy, when the discs became swollen a second time as the result of the meningitis. The case reported this evening belongs to the examples of partial atrophy, or, rather, of post-neuritic atrophy with preservation of good vision, perfect upon the right side and one tenth of normal upon the left.

With the reappearance of the neuritis, associated with symptoms of renewed intracranial disease, there was blindness, affecting not only the eye which had previously possessed poor vision, but also the one which up to this time had not suffered depreciation of sight. It is interesting in this connection to recall an observation sometimes made, that after the subsidence of the neuritis without much impairment of vision, there may be partial or entire loss of sight from renewed intracranial mischief without marked change in the ophthalmoscopic appearances.[1] In this case, on the contrary, the loss of vision is associated with great change in the ophthalmoscopic appearance, viz., double optic neuritis.

Another interesting phenomenon in our case is the following: In March, 1893, although the papillitis of the left eye was considerable (the swelling being + 6 D), the vision was normal. Three months later the vision in that eye had sunk to one tenth of normal, while that upon the opposite side had fallen to one half, and each macula now presented appearances not unlike those seen in albuminuric retinitis. These macular changes increased in the left eye, but remained stationary, or practically so, upon the right side. Gradually the vision of this eye, namely, the right, improved to the normal standard, while upon the left side, the vision, after having sunk as low as $\frac{3}{200}$, regained the $\frac{20}{200}$ level and remained there without further change until the reappearance of pronounced intracranial mischief, when the blindness became absolute, affecting equally the left and the right eye.

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[1] Gowers, *Medical Ophthalmoscopy*, second edition, Appendix, p. 292, Case 17.

Ophthalmoscopic appearances closely resembling those seen in albuminuric retinitis have been noted not infrequently in connection with cerebral tumor, and the plate of Mr. Edmunds,[1] illustrating this phenomenon is a familiar picture. If we may judge from a number of clinical experiences, and at least two post-mortem examinations, when these macular changes are pronounced and the papillitis is accompanied with the appearance of considerable mechanical congestion, the tumor is likely, other things being equal, to be found in the cerebellum, a point that is dwelt upon by Mr. Marcus Gunn in his paper on changes in the macula.[2] The early blindness which accompanies the optic neuritis associated with cerebellar neoplasms, is a clinical fact too well established to need description here.

It seems likely that in this case the original lesion, probably a glioma, was situated beneath the area exposed by the surgeon's trephine; that in the course of the months which have elapsed since that operation and the present time this tumor has extended, the extension having been preceded by a period of relief, both from headache and other symptoms of intracranial mischief, and in this extension it is not unlikely that the cerebellum has become involved.[3]

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[1] *Transactions of the Ophthalmological Society of the United Kingdom*, 1884.

[2] *Trans. Eighth International Ophthalmolog. Congress*, p. 79.

[3] J. Walter Park (*Annals of Ophthalmology and Otology*, January, 1895) describes an interesting case of acute middle-ear inflammation associated with two attacks of double optic neuritis, the interval between the attacks being a little more than a year. The condition of the optic nerves between the attacks is not stated. The patient made an excellent recovery in all respects, the final vision being $\frac{15}{50}$ in the right eye and $\frac{15}{70}$ in the left.

THE PARALLAX TEST FOR HETEROPHORIA.[1]

By D. A. DUANE, New York.

In the summer of 1889 I published in the *New York Medical Journal* a description of what I denominated the parallax test for insufficiencies. Since that time a continuous experience has convinced me that in this test we have a method which is not only among the most satisfactory of all that we possess, but also in some cases succeeds when the other tests fail or are self-contradictory. As the test is not generally known, and as the only detailed account of it appeared in a general and not a special medical journal, it seems not without utility to rehearse briefly the method of its performance and the principles upon which it is based.

To conduct the test, we place the patient in the primary position, with head erect and eyes directed straight forward or slightly below the horizontal plane (this latter especially in making the test for near points). The object of fixation, which should be twenty feet distant, may be a candle-flame, but preferably is a white spot, 1 or 2 *cm* in diameter, upon a dull black surface of some considerable extent. By this arrangement all danger of projecting the image upon a surface beyond is done away with, and the chance of a confusion with surrounding objects is prevented. The patient's gaze being directed fixedly at the spot, a card is placed before one eye, and passed alternately from that to the other,—the patient being at the same time asked whether the spot appears to move, and, if so, in what direction. If it remains perfectly stationary, there can have been no deviation behind the card, and the position of fixation of both eyes is perfect. If, however, the spot moves, it must occupy a different position as seen by the two eyes; *i.e.*, there is really a diplopia present which our method of observation has unmasked. Thus if, on uncovering the left eye, the object (which was previously seen by the right eye and is now seen by the left) appears to move to the patient's left, there is really a homonymous diplopia (homonymous parallax), which differs from ordinary diplopia only in the fact that the two images are seen alternately instead of at the same time; if the object seems to move to the right, there is crossed diplopia (crossed parallax); if the object moves down, the eye must have been higher behind the screen (left hyperphoria, left parallax); and if the object moves up, the left eye must have been lower behind the screen (right hyperphoria, right parallax). In order to determine the amount of this alternate diplopia, we place prisms of the appropriate direction and strength before one eye until the movement is abolished. Thus, supposing that when the left eye was uncovered the object seemed to move down and to the left (homonymous and left parallax, indicating a condition of hyperesophoria), two prisms are placed before this eye, with their bases respectively down and out, and increased in strength until the movement has become nil. The strength of the prism having its base down will measure the degree of hyperphoria, and that of the prism having its base out will indicate the degree of esophoria present.

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[1] Paper read before New York Academy of Medicine.

For near points the test is made in the same way, a small dot on a rather large card being employed, and the movement of the dot upon the card (and not of the card itself projected against some distant object) being observed.

Such being the nature of the test, and such the simple means by which it is carried out, it remains to state the reasons for employing it at all. This is requisite, for we have already a number of methods of measuring heterophoria, and the question may naturally be raised why we should multiply tests when enough exist already for the purpose in hand. The answer to this is that the complexity of the problem presenting itself for solution is often such that it is desirable to get confirmatory evidence of every kind before deciding, and, moreover, the other tests, while indicating the kind of heterophoria that is present, not infrequently give an erroneous impression as to its amount. Hence it is useful to have an additional source of information to supplement and check those which we have already; and it only remains to inquire if this method that we have described answers the demands that we can rightly make of it.

Of any test thus put forward we must ask how far it fulfils the requirements of applicability, precision, accuracy, and utility. As regards the first requirement, I may say that in my hands the test has proved to be almost universally applicable. There are very few persons who cannot with a little patience on the part of the examiner be got to recognize the displacement of the image, and to indicate its direction with accuracy. If the displacement is not recognized at once, it will in most cases be noticed after the patient's attention has been called to the artificial parallax produced when a prism of 2° or 3° is placed before the eyes. That is, the prism by exaggerating the parallactic displacement has forced it upon the patient's attention, and has made him recognize in what it consists; and he is then able to say whether a similar movement does or does not exist when there is no prism before the eye.

As regards precision the test leaves nothing to be desired. A prism of ½° suffices to produce or to neutralize quite a decided lateral parallax; and a vertical movement, corrected by a prism of even ⅛°, is clearly noticeable.[1]

As regards accuracy, I have become convinced from the constancy of the results which the method affords that it is one of the most reliable of all that we have at our command. And this, in fact, constitutes its reason for existence; establishes its claim to be used with, and sometimes in preference to, the other methods of determining insufficiency. A word upon this point will make this statement clear.

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[1] This precision makes it useful in determining whether a glass that the patient is wearing has or has not a prismatic element, and whether it is centered or not. If, for instance, a patient, who has no hyperphoria, shows, with his glass on, a vertical parallax, it proves either that the glass has a prismatic element in it, or is faultily adjusted (decentred) so as to produce a prismatic effect.

All the subjective tests for insufficiency depend in some way or other upon the production of diplopia. Usually this is effected by prisms, either applied roughly in the trial-frame, or with more precision in some one of the numerous varieties of phorometers. But the difficulty with the use of prisms has been that when two images of similar character are presented to the patient's notice, and he is required to state whether the two are or are not in the same vertical or horizontal line, he often unconsciously tries to bring them into line, and thus conceals an insufficiency which actually exists; or if the prisms are rotated, and the position of the images thus displaced, he instinctively tries to keep the images in line, and thus simulates an insufficiency which does not exist. This is often seen with the phorometer, and vitiates the findings of that otherwise most accurate instrument. Thus, when testing for near points, we are apt to find an exophoria, which increases almost indefinitely, although it is often quite certain that no such tendency to outward deviation actually exists. And while there are ways of obviating this objection that suggest themselves to the mind of the experienced examiner, they still constitute a source of uncertainty, particularly when the test is made for near points. Hence the attempt has been made to disassociate the images by giving one a different appearance from the other—*e.g.*, by converting a point of light into a luminous bar (Maddox rod), or into a blurred diffusion-circle (Stevens's spherical lens, with small aperture). But, as far as my experience goes, the impulse towards bringing two dissimilar images into line is almost as strong as when the images are alike. I believe that this is particularly the case with the Maddox rod. On account of this inaccuracy sometimes attaching to the subjective tests, some observers have preferred to rely solely upon objective methods, and have fallen back upon the old exclusion-test—*i.e.*, that in which the observer takes note of the deflection of one eye behind a screen, while the other eye is performing fixation. Now the parallax-test is nothing but the subjective correlative of the objective exclusion-test, and is superior to the latter, both in being far more delicate and in admitting of precise measurement. Like the exclusion-test, it may be applied while the patient is looking in any direction whatever, and thus a difference in the amount of deflection in various parts of the field of fixation may be observed and estimated.[1] Moreover, the results are stabile, and when once the deflection is corrected, any further attempt at correction with prisms will produce a deflection in the opposite sense, so that there is not in this test, as in some of the others, a tendency on the part of the patient to overcome the prisms, and thus simulate an insufficiency which does not exist. In other words, the two images, being seen alternately, there is not the same impulse to force them together, as obtains in the other tests, where the two images are seen simultaneously. Hence it will often be found that when the phorometer indicates an insufficiency of the interni, or exophoria, of anywhere from 5° to 10° for near, the parallax test will show constantly a deviation outward of not more than 1°; and corresponding to this small deviation it will be seen that the eyes remain steady behind the screen in performing alternate fixation.

Having used the test now for eight years, and upon a great number of cases, I am thoroughly convinced of its utility, and desire to recommend it to those who busy themselves with the consideration of anomalies of the ocular muscles. And whatever may be thought of the rôle that these anomalies play in producing direct or reflex disturbances, it is at least important that the data upon which our judgment of this difficult question is based should be as precise and as free from error as possible.

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[1] I have already (ARCHIVES OF OPHTHALMOLOGY, xxiii., p. 71) signalized the importance of observing this difference, which may give us a clue as to the nature and etiology of the heterophoria.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD QUARTER OF THE YEAR 1894.

By Dr. ST. BERNHEIMER, in Vienna ; Prof. C. HORSTMANN, in Berlin ; and Dr. P. SILEX, in Berlin ;

WITH THE ASSISTANCE OF

Dr. S. M. Burnett, Washington ; Dr. Dantone, Rome ; Dr. Herrnheiser, Prague ; Prof. Hirschmann, Charkow ; Dr. P. von Mittelstädt, Metz ; Dr. Sulzer, Geneva ; Dr. Werner, Dublin ; Dr. C. H. A. Westhoff, Amsterdam ; Dr. Schiötz and Dr. Ole Bull, Christiania ; Dr. R. Greeff, Berlin ; Dr. Deus, Berlin ; and others.

Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS. STATISTICAL REPORTS.

152. 35th Annual Report of the Hospital for Eye Diseases of the Netherlands. Utrecht, 1894.

153. Tscherning. *The Ophthalmological Works of Thomas Young*. Translated into French and edited by Tscherning. Preceded by a portrait of Young, a eulogy by Arago, and a preface by Javal. Pp. 248, with figures and 3 plates. Höst & Son, Copenhagen.

154. Bellarminoff. Report on the work done by the mobile ophthalmological departments. *Wratsch*, 1894, No. 28–29.

155. Van Leersum. Vertical chirography. *Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde*, ii., p. 377, 1894.

156. Czermak. *Operations on the Eye.* Parts 6 and 7. Carl Gerold, Vienna, 1894.

157. Fick. *Text-Book of Ophthalmology and Ophthalmoscopy.* With 157 figures, some colored, pp. 486. Veit & Co., Leipsic.

158. Standish, Myles. Report of the ophthalmic division of the Carney Hospital, Boston, for 1893.

159. Argyll–Robertson. The contributions of ophthalmology to general medicine (Presidential address). *Trans. Ophth. Soc. Un. K.*, xiv., p. 1.

In the Utrecht Hospital (152) 4221 patients were treated, 504 being in-patients, and 519 operations being done, 91 extractions, 50 enucleations, 89 tenotomies, and 6 advancements.

The ophthalmological works of Thomas Young (153) are not readily accessible, as the originals are scattered through the physiological transactions, and Peacock's book of the miscellaneous works of Thomas Young has not been extensively circulated. Tscherning's undertaking to render these works accessible to the general public is praiseworthy. May the wish expressed in the preface by Javal, that this book may be the starting-point for new labors in physiological optics, be fulfilled.

Sulzer.

In various remote districts in Russia where physicians are few, oculists (154) are sent for the purpose of affording at least temporary relief and of performing operations. The number of patients so treated was 7691, and 1466 operations were made.

Hirschmann.

Van Leersum (155) regards the vertical handwriting as better in every respect than the slanting. It is easier to read, more quickly learned, better for the eyes, and does not necessitate unfavorable postures. Westhoff.

The 6th and 7th parts of Czermak's (156) excellent work have appeared. Like the preceding parts these are distinguished by the lucid treatment of the material in hand.

Notwithstanding the number of good text-books, new ones are constantly appearing without there being a palpable need for them. Fick's (157) book is short and clear, and contains very good figures, many of which are colored.

At the Carney Hospital (Dr. Standish) (158) there were registered 1810 new cases in 1893. Fifteen extractions are tabulated, mostly by the combined method ; the others with a buttonhole in the iris ; no loss. BURNETT.

This address (159) contains remarks on the following subjects : Errors of refraction and nervous disorders ; the surgery of the tendon ; cocaine ; antiseptics ; counter-irritation, etc.

WERNER.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

160. BLOCK. A case of cyclopia. *Weekblad v. h. Nederlandsch Tijdschr. v. Geneeskunde*, pt. ii., p. 409, 1894.

161. BACH. On the micro-organisms of the conjunctival sac, their natural and artificial growth, and on the antiseptic value of eye-salves. *Graefe's Arch. f. Ophth.*, vol. xl., 3, pp. 130–220.

BLOCK (160) reports the case of a well-developed infant which showed only cephalic deformities and lived five hours after birth. The upper eyelid had no lachrymal point and was notched in the middle. The lid margins were provided with lanugo hairs. The lower lid evidently had been formed from the growing together of the right and left eyelids, and a notch showed the point of previous junction, and here lachrymal points were visible. When the lids were separated an eye not differing perceptibly from the normal was seen exactly in the middle of the square orbit.

WESTHOFF.

BACH (161) made a great number of experiments in relation to the presence of bacteria in the conjunctival sac. A conjunctiva of normal appearance was repeatedly tested for bacteria, and then the effect of winking and of the tears was studied, and he endeavored to find whether it was possible to render an infected conjunctival sac sterile, and what procedure would best accomplish it. For the latter purposes the disinfectant action of the eye-salves most in use was tested. The more important conclusions are as follows : 1. The mechanical cleansing of the conjunctival sac with the simultaneous irrigation with physiological salt solution is preferable to washing the sac with irritating antiseptics. 2. The cleansing of the conjuctival sac even when it appears normal is necessary before an operation. 3. Bandaging the eye favors the development of germs, and those present multiply. All microbes however do not find favorable conditions, and the influence of the tears must be taken into account. 4. Since cultures from the conjunctival sac of an eye which had been bandaged two days, were often sterile, it is proven that the sac can be made sterile. The course of healing is generally but little dependent upon the quantity of germs in the sac, but depends rather upon the technique and the operator, the course of the operation, the patient himself, and the condition of his circulatory apparatus. 6. An infection of the wound in cataract extraction can scarcely occur if proper precautions are taken.

The bichloride-vaselin salve (1:3000), and the 2 % silver and copper salves even in combination with an aqueous menstruum like the tears, have strong disinfectant properties ; the yellow salve, less. These salves are permanently and almost absolutely sterile. If the sac was not previously specially infected, it is in most cases possible to sterile the sac and even the lid margin by using bichloride-vaselin for twenty-four to forty-eight hours. If the plate shows innumerable colonies of staphylococci, the sac will not be rendered sterile even when the bichloride-vaselin has been used six to eight times in the forty-eight hours.

III.—INSTRUMENTS AND REMEDIES.

162. ROGMAN. Thioform in ocular therapeutics. *Flandre médic.*, Aug., 1894.

163. NICATI. Esthesiometry and photometry. A note in regard to the visual chart of the author. *Arch. d'opht.*, xiv., 5, p. 297.

164. GALEZOWSKI. Double spectacles with superimposed lenses for the use of aphakic patients and hyperopes. *Rec. d'opht.*, 1894, p. 525.

165. ANTONELLI. The Javal ophthalmometer employed for exophthalmometry and ophthalmostatometry. *Arch. d'opht.*, xiv., No. 9, 1894.

166. SIKLOSSY. On subconjunctival injections of bichloride. Report of a paper read before the twenty-seventh meeting of Hungarian physicians and natural scientists. *Wiener med. Wochenschr.*, 1894, No. 45.

167. DEUS. A new electric ophthalmoscope lamp. *Deutsche med. Wochenschr.*, 1894, No. 28.

168. PRINCE. The inclinometer in ophthalmology. *Ophth. Review*, xiii., p. 227.

169. Landolt. An ophthalmotrope. *Trans. Ophth. Soc. Un. K.*, xiv., p. 256.

170. McGillivray. The therapeutic value of ice in ophthalmic surgery. *Ophth. Review*, xiii., p. 294.

171. Bokenham. On benzol-pseudo-tropeine or tropa-cocaine. *Trans. Oph. Soc. Un. K.*, xiv., p. 234.

172. Howe, Lucien. Orthochromatic plates for photographing the interior of the eye. *Ibid.*, p. 251.

Rogman (162) has obtained favorable results in the treatment of purulent keratitis with thioform, which is used in the same way as iodoform. Sulzer.

Galezowski (164) has devised a spectacle frame with a second clip allowing an accessory glass to be placed in the frames in front of the stationary glass. Presbyopic, hyperopic, and aphakic patients can thus dispense with the frequent changing of their glasses. Sulzer.

Antonelli (165) used Javal's ophthalmometer for measuring the exophthalmus, the distance between the eyes, the difference in height, the deviation of one eye, and the extent and rapidity of nystagmic movements. The correct position of the ophthalmometer is obtained by means of a special apparatus, which is described in the original. In a case of pulsating exophthalmus he measured the extent of the exophthalmus, and found that even when the eye sank back in the orbit it still projected 2 *mm* anterior to the other eye. In congenital height differences, which occur with asymmetry of the skull, the author found the lower eye almost invariably hyperopic and astigmatic, a condition which, in his opinion, is of importance in the development of squint. v. Mittelstädt.

Siklossy (166) has used subconjunctival injections of bichloride in fifty-six cases of acute blennorrhœa with corneal complications, and in numerous cases of acute trachoma with corneal ulcers and pannus, and has obtained quick and good results with this method, which is not so painful as it is said to be.

Herrnheiser.

This is a graduated, flat, metal ring (168), with a strongly marked white line projecting towards the centre, and serving as an indicator. It can be used for finding the inclination of the shadow in retinoscopy, the position of the axis of a cylinder, the position of an obliquely elongated image, etc. Werner.

This is a very simple and useful instrument (169) for demonstrating ocular movements, diplopia, etc. WERNER.

McGILLIVRAY (170) recommends *continuous* cold in the early stage of eye injuries by means of iced compresses for a day or two. The cold constricts the blood-vessels and so prevents exudations and diapedesis ; it lessens pain by preventing pressure on the nerves, and also by paralyzing them, and it also impairs the activity of pathogenic micro-organisms. WERNER.

BOKENHAM (171) finds that tropa-cocaine acts more rapidly than cocaine, but does not cause mydriasis, cycloplegia, nor vascular changes, and is less poisonous. WERNER.

HOWE (172) showed photographs taken by the method described by him in the *Trans. of the American Ophth. Soc.*, vol. iv., p. 568. Plates for the purpose should be orthochromatic and as rapid as possible. WERNER.

IV.—ANATOMY.

173. KESSLER. The perichoroidal space in its relation to the lymph circulation in the eye. *Oogheelkundige Verslagen en Bijbladen.* Utrecht, 1894, p. 199.

KESSLER (173) found that Fontana's spaces and the loose tissue between the ciliary muscle and sclera form the beginning of the perichoroidal space, which is a fairly large lymph space furnishing a passage for fluid from the anterior chamber toward the perichoroidea, Schlemm's canal, and Leber's plexus. In all eyes with subnormal tension, the perichoroidal space becomes larger and may extend back to the optic nerve. Eyes with cyclitis are favorable objects in which to study the chief directions of the lymph currents, after the leucocytes have been stained. If the perivascular sheaths of the venæ vorticosæ can be considered the passages of exit for the fluid in the perichoroidal space, the continuous movement of the eyes would be an excellent means of conducting the lymyh through the loose tissue of Tenon's capsule toward the orbital tissue from which it can be readily carried off.

WESTHOFF.

V.—PHYSIOLGY.

174. TEPLIACHINE. Studies on the secretory nerves of the lachrymal gland. *Arch. d'opht.*, xiv., No. 7, 1894.

175. PARINAUD. The sensibility of the eye to spectral colors ; functions of the retinal elements and the visual purple. *Annal. d'Ocul.*, cxii., p. 228.

176. KOENIG. On the visual purple in man and its function in vision. *Sitzungber. der K. preuss. Akademie d. Wissenschr. zu Berlin*, p. 577.

177. SATTLER. Investigations on the question of the existence of an external accommodation from muscular pressure. *Graefe's Archiv*, xl., 3, p. 239.

178. v. HIPPEL. On total congenital color-blindness. *Festschrift for the 200th Anniversary of the University of Halle.*

179. HITSCHMANN. On the dream-life of the blind. *Zeitschr. f. Psych. u. Physiol. d. Sinnesorg.*, vii., parts 5 and 6, p. 387.

180. MARBE. On the doctrine of visual sensations resulting from successive excitations. *Inaug. Dissert.* Freiberg, 1893.

181. STERN, L. W. The perception of motion by means of the eye. *Zeitschr. f. Psych. u. Physiol. d. Sinnesorg.*, vii., parts 5 and 6.

182. STERN, L. W. The perception of changes in illumination. (Supplement), *ibid.*

183. v. HELMHOLTZ. *Handbook of Physiological Optics.* 2d ed. 8th part. Voss, Hamburg and Leipsic, 1894.

184. GRIFFITH. Criticism concerning recent views as to the secretory function of the ciliary body. *Ophth. Review*, xiii., p. 247.

TEPLIACHINE (174) reviews the views previously held in regard to the secretory nerves of the lachrymal glands and endeavors to solve the question by his own experiments, which led him to the following conclusions:

The view that the facial is the secretory nerve is untenable. The secretory fibres of the lachrymal and subcut. malæ nerves run intracranially in the trigeminus trunk and take part in the reflex secretion of tears and that caused by psychic influences. The normal and constant secretion of tears is not brought about reflexly by irritation of the anterior segment of the eye, and does not depend upon the secretory fibres of the lachrymal nerve, but is produced by the cervical sympathetic. The author observed two cases of complete unilateral facial paralysis in which no lachrymation could be produced reflexly, but there was also paralysis of the fifth nerve with anæsthesia of the cornea, conjunctiva and skin of the face. Notwithstanding this, the eye of the paralyzed side was more moist than the other, and the secretion of tears continued. v. MITTELSTÄDT.

PARINAUD (175) by means of a spectrum apparatus had made a series of experiments on the perception of the spectral colors. The increased sensibility of the adapted retina differs with the different wave lengths. It is 0 for red, and gradually increases toward the blue end of the spectrum. The maximum of intensity in the spectrum for the adapted retina is at D, and for the unadapted eye is near E. The increased sensibility for a given wave length is only as regards light intensity, and not color perception.

Koenig's (176) careful experiments with monochromatic light and his mathematical calculations prove that different colors pass into different depths of the perceptive layers of the retina so that the different colors are not perceived in the same substance. Between the colors of shortest and longest wave length, there is a difference of 0.0786 *mm*, indicating that the perception of light of greater wave length takes place farther outward in the retina than the perception of light of shorter wave length. These facts do not agree with the color theories of Hering, Ebbinghaus, Donders, Wundt, and Franklin, while they are in accord with the Young-Helmholtz theory.

v. Hippel's (178) careful examination of the eyes of a woman of twenty-seven with congenital total blindness furnishes a new support for the Hering theory.

Shortly after Helmholtz's (183) death, the eighth part of the second edition of his *Physiological Optics* appeared, which will be completed by A. Koenig. This part contains the doctrine of contrast sensations, of the different subjective sensations, of visual perceptions in general, and of ocular movements.

Griffith (184) gives a detailed account of the structure of the ciliary region partly from the study of bleached sections. (He recommends bleaching the tissue *en bloc*, with a solution of chlorate of potash and hydrochloric acid, in order to avoid the brittleness of bleached sections.) He arrives at the conclusion that the aqueous humor is secreted by the plicated surfaces of the ciliary body and not by the glands of the ciliary body. True glands do exist in the pars non plicata, but contrary to Collins's opinion they are very few in number (two or three in a section). The complicated folding of the surface of the ciliary processes, combined with obliquity of section, may produce appearances like glands ; again, foldings of the epithelium produced by contraction of the ciliary muscle may be mistaken for them. There are no real glands in the ciliary region of albino rabbits, yet they have aqueous humor. The few glands which do really exist in human eyes may probably control the amount of pigment in the eye. The theory of glandular hyperplasia in cyclitic membranes, as enunciated by Alt, and accepted by Collins, he considers absurd and contrary to general pathology. He looks upon these so-called gland proliferations as epithelial foldings due to cicatricial contractions, or as newly formed lymph channels congested with leucocytes. Against Nicati's view, that the aqueous humor is formed by exudation from the choroidal chorio-capillaris, which is contained between two impermeable membranes, the lamina vitrea internally and the intervascular membrane of Sattler, (tapetum) externally, the author points out that extensive choroidal disease does not alter the intraocular tension, while cyclitis does ; he also asks, Is the membrane of Bruch really impermeable ? And does the tapetum exist in man ?

WERNER.

Sections VI.–XI. Reviewed by DR. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

185. KOTOSCHOWSKY. Sciascopy. *Wajenno medic. Journ.*, Apr., 1894.

186. KATZ. On the determinations of refraction by means of sciascopy. *Arch. f. Augenheilk.*, xxix., p. 142.

187. BORDIER. The determination of the acuteness of vision in ametropic eyes by means of Badal's optometer. *Arch. d'opht.*, xiv., 9, p. 562.

188. MARTIN. On monolateral myopia. *Annal. d'Ocul.*, cxii., p. 1.

189. MASSELON. On nasal sclerectasia in myopia. *Annal. d'Ocul.*, cxii., p. 20.

190. HORI. A contribution to the operative treatment of myopia of high degree. *Arch. f. Augenheilk.*, xxix., p. 142.

191. FUKALA. The indications and contra-indications for operation in myopia of high degree. *Wiener klin. Wochenschr.*, No. 47, 1894.

192. ANDOGSKY and DOLGANOFF. Clinical notes on astigmatism and its correction by means of Javal's ophthalmometer. *Wratsch*, 1894, No. 36–37.

193. RISLEY. The results of treatment and the optical correction of ametropia in arresting the increase of myopia. ARCH. OF OPHTH., xxiii., p. 247.

194. SOUTHARD. The modern eye. *The Refractionist*, June, 1894.

195. SMITH, PRIESTLEY. On periodical testing of the eyesight in schools. *Trans. Ophth. Soc. Un. K.*, xiv., p. 220.

196. WRAY. The eyesight of children. *Ibid.*, p. 228.

197. CLARKE. The association of blepharitis and ametropia with analysis of 100 cases. *Ophth. Rev.*, xiii., p. 345.

KATZ (186) calls attention to the sources of error in sciascopy, which are present both when the macular region is tested (involuntary accommodation, narrow pupil), and when peripheric portions of the fundus are tested (excavation), using the peripheric less curved portion of the cornea, etc., and also the fact that the shadow does not reverse at a neutral point but in an interval whose centre corresponds approximately to the far point of the examined eye. The greatest accuracy is obtained: 1st, by throwing the light in a direction which makes the smallest possible angle with the visual axis of the examined eye; 2d, by using as source of light a round sharply outlined surface; 3d, by dilating the pupil of the examined eye and by making the perforation in the mirror small. HIRSCHMANN.

BORDIER (187) shows that for determining the actual acuteness of vision in ametropic eyes, Badal's optometer is the best adapted instrument, and he gives the necessary procedures for cases of axial ametropia and ametropia of curvature.

V. MITTELSTÄDT.

MARTIN (188) collected accurate clinical and ophthalmometric data of 180 cases of unilateral myopia, and criticised the theories of the production of myopia. Astigmatism, corneal inflammation, and trauma are important factors. SULZER.

Nasal sclerectasia (189) was mentioned in Masselon's *Ophtalmoscopie Clinique*, 2d edition, 1891. SULZER.

HORI (190) reports fifteen cases of myopia of high degree in which Schweigger in 1892–93 performed discission of the lens. The oldest patient was thirty-four. The myopia varied from 13–14 D. In three cases there was slight hyperopia after the operation, in three slight myopia, and in nine emmetropia. The acuteness of vision was more or less improved in all.

FUKALA (191), who has now performed discission for myopia in forty-four cases, recommends the operation only in persons under forty.

SOUTHARD'S (194) paper is based upon an examination of 1300 ametropic eyes. Divided generally, there were 1017 cases of all forms of hyperopia, and 283 cases of all forms of myopia. The ages ranged from five years (1 %) to seventy years (1 %), the largest number (23 %) being at twenty years—that at ten years being 8 %; only 57.61 % had vision of $\frac{20}{20}$. There was simple H in 58 %, simple M in 8 %, and astigmatism of all kinds in 33 %. Other valuable tables too lengthy to abstract are given. BURNETT.

P. SMITH (195) advocates the yearly testing of sight, with Snellen's types, without glasses, by a master, who should report cases with V L $\frac{6}{18}$. This method involves no expense, and would detect all errors except hypermetropia, which of itself would cause symptoms sufficient to attract attention. WERNER.

WRAY (196) insists on surgeons examining the parents' refraction for prognosis and treatment of the children's eyes. Rapid disappearance of hypermetropia should be carefully noted.

WERNER.

In 100 cases of blepharitis CLARKE (197) found 84 % of hypermetropia. Out of 200 eyes 194 were hypermetropic or astigmatic. In 96 cases of H. astig., 72 were not more than 0.5 D astigmatic. Average age seventeen. Only one patient was emmetropic, whereas in 100 eyes examined by W. Dodd, without disease, 20 % of emmetropes was found. WERNER.

VII.—LIDS.

198. ZEILENDORF. On congenital coloboma of the lid. *Wiener med. Wochenschr.*, 1894, No. 34.

199. KRISCHEWSKY. On the etiology of congenital coloboma of the lid. *Verf. d. Phys. med. Ges. zu Würzburg*, 1894, No. 5.

200. BERNHARDT. A contribution to the subject of the peculiar synchronous movement of the paretic upper lid in unilateral congenital ptosis. *Neurolog. Centralbl.*, 1894, No. 9.

201. HILLEMANNS. Peculiar synchronous movement of the upper lid of an eye with coloboma of the optic nerve. *Ibid.*, p. 388.

202. MÜLLER-KAUNBERG. Peculiar synchronous movement of an upper lid with ptosis, with movement of the lower jaw. *Jahresbericht d. Frohlich'schen Augenklinik in Berlin*, 1894.

203. Stephenson. Note upon a form of congenital trichiasis. *Trans. Opth. Soc. Un. K.*, xiv., p. 13.

204. Watson, Spencer. A new operation for trichiasis and distichiasis, with a case. *Ibid.*, p. 17.

Zeilendorf (198) reports two cases of congenital coloboma of the lid. In the first case there was a V-shaped defect in the lower lid almost under the caruncle ; in the other case there was a similiar defect in each upper lid.

Krischewsky (199) believes that the congenital coloboma of the lids may be the consequence of the imperfect junction of the frontal and the superior maxillary process ; thus he would explain the triangular defects of the lower lid.

Bernhardt's patient (200), a boy of nineteen, with congenital right ptosis, raised the lid when the lower jaw was dropped or when it was moved to the left.

Similar cases were observed by Hillemanns (201) and Müller-Kaunberg (202).

Out of fourteen cases seen by the author (203) it was bilateral in nine. The displacement of the cilia, which occurs chiefly towards the inner canthus, is due to a fold of skin which rises up parallel to the lid margin, hides the intermarginal portion from view, without inverting it, and thus presses the eyelashes against the globe, causing irritation and corneal troubles. The author believes that defective development of the tarsus, and not any abnormality of the orbicularis is the cause of it.

(I have seen two instances of this condition in infants, and in one of these it disappeared under chloroform, a fact which would seem to support the second theory.—L. W.). Werner.

This is a modification of Watson's (204) older operation. An oval flap of skin, above the lid margins, is left adherent by its under surface and then pushed through a " button-hole " and fastened in the intermarginal incision. Werner.

VIII.—LACHRYMAL APPARATUS.

205. Cuenod. Two cases of dacryocystitis with ozæna. Bacteriological examination. *Arch. d'opht.*, xiv., 9, p. 495.

206. Dianoux. Tumors of the lachrymal gland. *Annal. d'Ocul.*, cxii., p. 81.

Cuenod (205) in two patients with ozæna having acute dacryocystitis, found Lowenberg's ozæna bacillus in pure culture without other pyogenic microbes in the pus of the tear sac.

Dianoux (206) saw within a short interval four cases of tumor of the lachrymal gland. The first was extirpated, recurred, and finally necessitated evisceration of the orbit. Six months after the last operation there had been no recurrence. The patient was thirty-six years old. In the second patient, aged twenty, there was recurrence fifteen months after the extirpation of a completely circumscribed tumor. In the other two cases the affection was tubercular. The two tumors removed in the second case had a similar structure, that of a polymorphous epithelioma.

Sulzer.

IX.—MUSCLES AND NERVES.

207. Schmidt-Rimpler. On binocular vision in squinting individuals before and after operation. *Deutsche med. Wochenschr.*, 1894, No. 44.

208. Landolt. On strabotomy. *Arch. d'opht.*, xiv., 8, p. 474.

209. Panas. Paralyses of the motor muscles of the eye after lateral pressure on the skull. *Ibid.*, p. 465.

210. Valk. The dynamics or power of the interni. *Med. Record*, July 21, 1894.

211. Hall, Geo. P. A contribution to the study of the insufficiencies of the ocular muscles, with measures directed to their relief. *Trans. Texas State Med. Soc.*, 1893.

212. Frost. Nystagmus in a man who has worked in a bad light for eighteen years. *Trans. Ophth. Soc. Un. K.*, xiv., p. 245.

213. Thomas, C. H. Three cases of strabismus with anomalous diplopia. An original and an acquired fixation spot in the same eye. *Ophth. Review*, xiii., p. 281.

214. Williams, R. The operation of advancement in squint. *Ibid.*, p. 377.

215. Taylor, Johnson. A case of probable disease of the lenticular ganglion. *Ibid.*, p. 290.

According to Schmidt-Rimpler (207) a squinting individual ordinarily only sees the image of the eye to which he directs his attention. It is therefore advantageous to practise the squinting eye before the operation in order to facilitate binocular vision after the operation.

Landolt (208) on the ground of increased experience recommends, as before, advancement as the chief procedure for the correction of squint. He makes this operation, in cases of insufficiency and ordinary squint, on one eye or both, and when necessary with resection of a portion of the tendon. Only in cases of excessive deviation does he make a careful tenotomy in addition. The disadvantages of tenotomy—restriction of mobility, sinking of the caruncle, and protrusion of the ball—are impossible in advancement which corrects the squint in the entire field of fixation. An excessive effect need not be feared, nor a vertical deviation, nor a meridional torsion. v. MITTELSTÄDT.

In a man whose skull had been compressed laterally, probably causing fracture of both petrous bones and the sphenoid, PANAS (209) found paralyses of all the ocular muscles the right auditory and the left facial nerve. There was bleeding from the ears and nose immediately after the operation. Later there were severe headache and a sero-purulent discharge from the right ear, and two weeks after the injury the paralyses appeared. By literal compression of the head of a corpse Panas was able to produce a double fracture of the temporal bone which would have explained the symptoms found. In another experiment, by compressing the skull in the temporal region, he obtained a basal fracture of the middle fossa which extended toward the sphenoidal fissure, and passed through the outer wall of the left orbit and under the frontal portion of the sphenoid and the optic foramen on the right side. The temporal bones were simply displaced. In both experiments, the roof of the skull remained uninjured. Panas reports also a case corresponding to the second experiment, in which after a fall on the head there were hemorrhage from the left ear, bilateral subconjunctival hemorrhage, and paralysis of the abducens and optic nerves of the left side. Panas concludes that the majority of the ocular muscle paralyses after skull injuries are due to fracture of the base. The abducens is the nerve most frequently affected because of its nearness to the bone. The paralysis is due either to direct injury of the nerves, or to the presence of blood, or inflammatory exudation. v. MITTELSTÄDT.

VALK (210) has found, as the result of his experience and examinations in more than 50% of his asthenopic cases, an excess of power in the externi. He places but little reliance upon those methods of testing for insufficiency in which one retinal image is reduced by any means. He relies on the old prism test for ascertaining the actual power of the muscle—exercise has proved useful only in weakness of the interni. Where the insufficiency is well marked he does a partial tenotomy. BURNETT.

HALL (211) reports a number of cases where the lack of muscular balance was entirely independent of any refractive error; he has used gymnastic exercise, prisms, and tenotomies, all with success, in relieving the faulty condition. BURNETT.

FROST'S (212) patient worked at night washing cabs in a badly lighted yard. $V = \frac{6}{6}$. Nystagmus occurred on raising eyes above the horizontal line. Slight improvement on cessation of work. WERNER.

THOMAS (213) records two cases of divergent strabismus, in one of which there was *homonymous* diplopia before operation, while after it the diplopia agreed with the position of the eyes, $V = \frac{6}{6}$; H = 0.5 D. In the second case after operation, which diminished the amount of divergence, *homonymous* diplopia appeared. The last case, one of convergence, showed *crossed* diplopia after incomplete correction by operation. In all cases there was central fixation with each eye separately. WERNER.

WILLIAMS (214) makes a horizontal incision in the conjunctiva over the muscle, and the suture is passed through the muscle from side to side at right angles to the direction of its fibres.

WERNER.

TAYLOR'S (215) patient, a woman, aged forty-nine, with — 16 D, developed rather quickly dilatation and immobility of left pupil, with some proptosis and a little haziness of vision; there was neuralgic pain about the eye and down the nose. She had suffered some years before from pain and loss of power in one arm. The author accounted for the ocular condition by assuming that orbital cellulitis was present and damaged the lenticular ganglion.

WERNER.

X.—ORBIT AND NEIGHBORING CAVATIES.

216. MITVALSKY. On orbital thrombo-phlebitis. *Prager med. Wochenschr.*, 1894, No. 45.

217. BOERMA. A case of symmetrical lymphoma. *Graefe's Archiv*, xl., 4, p. 219.

218. MARTIN. Orbital tumors caused by frontal-sinus disease. *Annal. d'Ocul.*, cxii., p. 182.

219. BERGER. Varieties of the wall of the optic foramen. *Arch. d'opht.*, xiv., 9, p. 545.

220. GRIFFITH. Some cases of orbital growths. *Trans. Ophth. Soc. Un. K.*, xiv., p. 173.

221. Snell. Congenital cysts of the eyelids associated with anophthalmus or microphthalmus. *Ibid.*, p. 190.

222. Clarke. Pulsating exophthalmus (congenital). *Ibid.*, p. 202.

223. Green, E. C. A case of exostosis of the orbit; operation. *Ibid.*, p. 186.

224. Williams, R. Case of rapidly growing sarcoma of the orbit. *Ibid.*, p. 186.

225. Snell, S. Rapidly growing sarcoma in a child involving both orbits with secondary growths. *Ibid.*, p. 160.

226. Snell, S. Osteoma of the orbit. *Ibid.*, p. 169.

227. Power, H. Case of microcephalus and proptosis. *Ibid.*, p. 312.

228. Stephenson, S. Cellulitis following Mules's operation. *Ophth. Review*, xiii., p. 286.

Mitvalsky (216) examined two cases of orbital thrombo-phlebitis clinically, anatomically, and bacteriologically. The first case, in a man of fifty-six, was due to a gangrenous amygdalitis. Anatomically there was found first a hemorrhagic inflammation of the orbital veins, and later an œdema of the orbital connective tissue. In the fresh venous thrombus and also in the sections the Fraenkel-Weichselbaum diplococcus was found. In the second case the staphylococcus pyogenes was the cause of the infection.

Herrnheiser.

Boerma (217), in a woman of sixty-three, observed symmetrical orbital tumors pressing the lids and conjunctiva forward, which could be pressed back to a certain degree beneath the orbital margin. The tumors were removed with preservation of the ball, and proved to be lymphomata of the orbit.

Following empyema of the frontal sinus, Martin (218) observed swelling of the neighboring orbital tissue and almost complete loss of sight, with exophthalmus and displacement of the ball outward and downward. Besides the subcutaneous collection of pus there was a protrusion of the orbital wall of the frontal sinus. Catheterization and antiseptic injections cured the case completely, and the author does not favor trephining the sinus.

Sulzer.

Berger (219) gives drawings of various frontal sections through the optic foramen, showing the varying thickness of the wall of the sphenoidal sinus adjacent to the optic nerve. In places it is very thin on each side, again thin on one side and thick on the other. At times the wall is broken so that the mucosa of the sinus must lie in contact with the sheath of the optic nerve, and thus is to be explained the affection of the optic nerve in diseases of the sinus. In 1888 Berger had collected twenty-three such cases verified by autopsy. Of the other neighboring cavities, only the ethmoid could also play a similar rôle in the production of optic-nerve trouble. v. MITTELSTÄDT.

GRIFFITH (220) records the following cases: 1. Fibro-sarcoma in a man aged thirty-five, removed without recurrence. 2. Exostosis in a girl aged fifteen. 3. Symmetrical orbital tumors, with motor and sensory paralyses of orbital region, improving under treatment. 4. Probable fibrous tumor; and, finally, two cases of malignant sarcoma, with fatal result after operation, which consisted in circular incision round orbital margin, and removal of contents of orbit. WERNER.

SNELL (221) records three cases, two occurring in the lower lid with apparent anophthalmus, and one in the upper lid with microphthalmus. In the last case, the child vomited when the cyst was tapped; it pulsated synchronously with the anterior fontanelle. Chemical examination of the fluid showed that it was not cerebro-spinal. All three cases recovered, the two first after removal of portion of the cysts, and the last after three tappings. WERNER.

In CLARKE'S case (222), the birth of the child was normal. In addition to the pulsation there was an intermittent blowing murmur. Optic disc atrophied, arteries small, veins dilated. The president mentioned two traumatic cases cured by electrolysis. WERNER.

In GREEN'S (223) case the exostosis was removed by the electromotor drill in about seven minutes, with success. WERNER.

In WILLIAMS'S (224) case, a man aged thirty-three, death took place within two months after the onset of the symptoms. When the eye was enucleated no distinct tumor could be felt in the orbit, but twenty-four hours later the growth had filled it up. WERNER.

SNELL'S (225) patient was aged four. A diffuse periosteal sarcoma affected the frontal bone, involving both orbits. WERNER.

In SNELL'S (226) case, a girl aged twenty-five, there was an osteoma of the inner wall of the orbit, and another in the nostril, both of which were easily removed, and were probably unconnected. WERNER.

In POWER'S (227) case the birth was normal, but the child lived only thirty days. The vertex and frontal region were drawn out into a blunt cone. The eyes projected forwards, owing to shallowness of the orbit, which was only 2.5 *cm* in depth. The roof was almost vertical, and the optic nerve ascended in contact with it.

WERNER.

Out of 30 cases in which STEPHENSON (228) did evisceration, with insertion of an artificial vitreous, this was the only case in which severe cellulitis occurred. The glass ball was removed, and the stump subsequently enucleated; the sclera was found to have undergone extensive hyperplastic changes. WERNER.

XI.—CONJUNCTIVA, CORNEA, ANTERIOR CHAMBER, AND SCLERA.

229. VENNEMANN. The surgical treatment of granulations. *Arch. d'opht.*, xiv., 7, p. 413.

230. GLEISER. The statistics of trachoma. *Annal. d. kais. Charkow'schen Universität*, 1894.

231. BLAAUW. Some considerations on the nature and treatment of trachoma. *Inaug. Dissert.*, Amsterdam, 1894.

232. TRUC. The general character and contagiousness of trachoma in the region of Montpellier. *Annal. d'Ocul.*, cxi., p. 328.

233. KRUCH and FUMAGALLI. Amyloid degeneration of the conjunctiva. *Annal. d'Ocul.*, cxii., p. 39.

234. SAULAY. Note on conjunctival papilloma. *Rec. d'opht.*, 1894, p. 543.

235. SCHIRMER. Cure of a cicatricial pterygium by corneal transplantation. *Deutsche. med. Wochenschr.*, 1894, No. 37.

236. WEYMAN. Conjunctival polypi and their relation to malignancy. *The Ophth. Record*, Sept., 1894.

237. HOBBS. The treatment of pterygia with the galvano-cautery. *Journ. Amer. Med. Assoc.*, Sept. 15, 1894.

238. JESSOP. Case of sloughing of both lids and the eyeball in an infant, with subsequent complete cicatrization. *Trans. Ophth. Soc. Un. K.*, xiv., p. 22.

239. HUTCHINSON. On school ophthalmia. *Ibid.*, p. 35.

240. RIDLEY. Some points on the etiology of trachoma. *Ibid.*, p. 24.

According to VENNEMANN (229) the follicles in trachoma are innocent and may disappear of their own accord, but as cicatricial shrinking follows in the hypertrophied and degenerated conjunctiva the surgical methods of treatment are not rational. The author therefore used the old procedure of Peters-Keining. After the use of cocaine the epithelium of the conjunctiva is removed with Demarres' scarificator, the conjunctiva is irrigated with 1:500 bichloride solution, and, the following day, rubbed with cotton which has been dipped in this solution. The third day a croupous membrane forms, which is a favorable sign. The results were good. Relapses occurred, but the treatment is not disagreeable and the patient readily consents to a second operation.

v. MITTELSTÄDT.

The total number of trachoma cases treated in the Charkow clinic (230) in the years 1867–1892 was 21,218, or 13 % of all the eye cases seen. The percentage of trachoma cases increased regularly from 7.8 % in 1867 to 16.4 % in 1892. A particular racial predisposition could not be made out. The percentage of severe, neglected, and complicated cases has steadily decreased from 59.2 % to 42.5 %. In the statistics of blindness for the years 1869–1873 trachoma was noted as the cause in 6 %.

HIRSCHMANN.

According to BLAAUW (231) trachoma is not contagious, but develops where hygienic conditions are unfavorable. A specific microbe has not been demonstrated and it is not probable that such exists. In typical trachoma there is no mucus and no pus. It is chiefly found in children under six. It is always chronic, and cases do not require isolation. WESTHOFF.

TRUC (232) sees in his clinic one hundred times more trachoma patients than in his private practice. Almost all the clinic cases are scrofulous. The purulent variety is rare, and the ordinary dry form is benign.

KRUCH and FUMAGALLI (233) came to the following conclusions. Amyloid degeneration of the conjunctiva is a local condition not dependent upon constitutional causes. The local excision of the diseased tissue improves the condition and may cure it, even when the excision is not complete. The amyloid substance is formed chiefly at the expense of the subconjunctival connective tissue, and does not originate from the vessels. It is probably of parasitic origin. SULZER.

In a case of large cicatricial pterygium involving two thirds of the cornea, complicated with extensive symblepharon, SCHIRMER (235) detached the pteryguim from the cornea and detached the conjunctival folds and cicatricial bands from the sclera. The defect above the sclera was covered with conjunctiva, and the denuded corneal surface was scraped smooth with a sharp spoon and filled in with flaps from the surface of a rabbit's cornea, which remained opposed and healed there. The mobility of the ball was preserved and only a small corneal leucoma remained.

JESSOP'S (238) patient was aged nine months and had ophthalmia neonatorum. WERNER.

HUTCHINSON (239) discusses at some length the question of the nature of a mild form of ophthalmia with development of follicles, and in a very few instances leading to granular ophthalmia. Contrary to the opinion of nearly all present who took part in the discussion, he believed this form of ophthalmia to be the precursor of trachoma (in no case was there any pannus), and as such required stringent observation, and regulation as to isolation, etc. Most of the speakers looked upon the presence of small follicles as a very common condition in children. Stephenson found them in 94 per cent. of children in schools in which there was no ophthalmia, and he was disposed to regard them as normal. Doyne thought that the author's cases were cases of muco-purulent conjunctivitis in subjects whose lids were in a follicular condition. WERNER.

RIDLEY (240) in a very interesting paper describes the histology of the normal conjunctiva of the fornix, and also of the same part when affected with trachoma. Foa's solution was used as a fixing agent. The normal conjunctiva possesses stratified epithelium, the cells being columnar below, then rounded, and finally squamous on the surface ; there are some goblet cells near the surface. The folded condition at the fornix produces on section the appearances described as Krause's glands. There is a variable amount of diffusely scattered lymphoid tissue, which also exists under other mucous membranes, especially around openings. In all forms of conjunctivitis this lymphoid or "sentinel" tissue is increased ; this increase is not a disease, but evidence of the reaction of the organism to an injurious agent.

The lymphoid tissue in trachoma differs from the same tissue in other diseases only in its amount and arrangement. It occurs as a diffuse infiltration of the subconjunctival connective tissue, and in the shape of round packets, occasionally encapsuled, covered only by epithelium, in which it causes projections and furrows. These lymph follicles resemble infective granulomata and contain epithelioid cells, but no giant-cells; they differ, moreover, from tubercle in containing healthy blood-vessels.

According to the above, granular ophthalmia is not necessarily due to the same cause in every case, but there may be a specific cause in certain cases. The epithelium is probably the seat of the irritant, and improvement takes place on destroying the epithelium. According to the author's investigations, the normal conjunctival sac contains the same micro-organisms as it does when trachoma is present. The changes in the epithelium include increased proliferation, deepening of the folds, so as, in some places, to form cysts, and increase in the number of goblet cells. Many of these so-called goblet cells are not really such, for they lie in the deepest layers, often parallel to the surface, and they stain differently: they may be protozoa. Amongst other things, the slight contagiousness of granular ophthalmia is against its being of bacterial origin. Werner.

241. Fuchs. On keratoplasty. *Wiener klin. Wochenschr.*, 1894, No. 45.

242. Critchett. A case of conical cornea treated by galvano-cautery without perforation of the cornea, and subsequent small iridectomy. *Trans. Ophth. Soc. Un. K.*, xiv., p. 73.

243. Griffith. Blood-staining of the cornea. *Ibid.*, p. 73.

244. Cowell and Griffith. A case of filamentous keratitis. *Ibid.*, p. 76.

245. Scott, Kenneth. A new method of treatment for vascularized corneæ. *Ophth. Review*, xiii., p. 348.

Fuchs (241) has done total keratoplasty in 30 cases; 2 with parenchymatous keratitis, 12 with true superficial corneal scars with adhesions of the iris, 8 extensive total ectatic scars from prolapse of the iris; 14 times he used a rabbit's cornea, 11 times that of an enucleated human eye, and several times the cornea of dogs or of fresh still-born infants. In 11 of the 30 cases the transplanted piece of cornea did not adhere. In no case did the transplanted piece remain transparent, it becoming opaque, as a rule, after a few days.

In Critchett's case (242) V was improved from Jaeger 8 at 3 inches to Jaeger 2 at 10 inches and $\frac{6}{18}$ — 9 Cyl.

Werner.

Griffith's (243) patient had discission performed; three months later there was pain, high tension, hyphæma, and smoky cloudiness of the cornea, except at periphery. On section, the substantia propria of the cornea was studded with small, round, yellow granules, highly refractive, insoluble in acids, alkalies, ether, alcohol, etc., stained only by eosin- and iodin- or methyl-green. As in cases recorded by Lawford and Treacher Collins, there was high tension with the hyphæma, and also detachment of the retina. Werner.

This case (244) presented the usual twisted filaments with bulbous extremities attached to the cornea. The bulbs stained with fluorescin. Werner.

Scott (245), with the aid of a lens, slits open the corneal vessels with a Graefe's knife. He finds this much better than the cautery. Werner.

246. Fage. Immediate and late results of scleral sutures. *Annal. d'Ocul.*, cxii., p. 262.

247. Collins, E. T. Case of a patient with congenital excess of pigment in the uveal tract, and pigmentation of the sclerotic in one eye, which late in life became the seat of a melanotic sarcoma. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 197.

Fage (246), after a considerable experience in suturing scleral wounds with fine catgut, recommends the procedure. The late occurrence of irido-cyclitis, when the eye is apparently cured, is seen after injuries in which septic bodies have entered the vitreous. Sulzer.

248. Snellen. Descemetitis. *Ophth. Review*, xiii., p. 259.

249. Coppey. A case of filaria in the anterior chamber of a human eye. *Arch. d'opht.*, xiv., 9, p. 557.

Snellen (248) refers to the various views as to the nature of this affection, *e.g.*, a disease *sui generis*, serous iritis, serous cyclitis, catarrhal inflammation of the glands of the ciliary body; he then records three cases in which there was k. punctata, with slight circumcorneal injection, and no other symptoms of iritis, except synechiæ, where dots of lymph formed on the iris. In each case paracentesis enables the author to examine a dot, which he found to consist chiefly of short bacilli, showing, as he believed, that this was a disease *sui generis*, caused by the bacilli.

Werner.

Coppey (249) observed a filaria in the anterior chamber of a negro infant, which six weeks before had come from Congo, and at that time showed two small nodules on the iris, which soon disappeared. A thread worm one half *mm* thick and three *cm* long was seen moving in the anterior chamber. The eye was injected, the aqueous was clear, but the pupil could not be illuminated. The anterior chamber is the rarest location in the eye for the filaria, and to find a living specimen there is exceedingly rare. In most of the sixty cases in which Firket examined the blood of inhabitants of Congo, one or two filaria embryos were found in every drop of blood, so that it seems probable that in this case the filaria has been carried into the eye by the blood current.

Sections XII.–XXII. Reviewed by Dr. SILEX.

XII.—IRIS.

250. Wicherkiewicz. Pseudo-gumma of the iris of traumatic origin. *Zehender's klin. Monatsbl.*, xxxii., p. 278.

251. Fourier. Syphilitic iritis. *L'union méd.*, March 1, 1894.

252. Mocheck. On miliary tuberculosis of the iris. *Wien. med. Wochenschr.*, No. 24–25, 1894.

253. Sandford. Three cases of tubercle of the iris. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 90.

254. Fergus. Note on peculiar ophthalmic conditions occurring in members of one family. *Ibid.*, xiv., p. 206.

Wicherkiwiecz (250) reports two cases in which a foreign body had produced the clinical picture of gumma of the iris, and he remarks that we have no sure criterion of gumma, tubercle, and granuloma. On the other hand, in the case of a foreign body, an existing syphilis may be overlooked. A case of the latter sort was cured by specific treatment.

Mocheck (252) observed three cases of tuberculosis of the iris in children. The two first died of tubercular meningitis. This was diagnosed in the third case also, but two years later the child was quite well. The features of miliary tuberculosis of the iris are as follows: The disease begins as a chronic iritis; there is little photophobia and the pain is slight. The moderate plastic exudation which usually blocks the pupil forms undetachable posterior synechiæ. There is always precipitation on the posterior surface of the cornea. The swelling of the tissue is irregular, and the new formations are richly vascular. In the later course of the disease the miliary tubercles are usually visible.

SANDFORD'S (253) first case was one of miliary tubercles of the iris in a phthisical lad aged sixteen, and ended in recovery. The second case was exactly similar, occurring in a phthisical girl aged nineteen. The third case was one of rapidly growing solitary tubercle in a child aged five with very tubercular antecedents. As it was regarded as a case of primary tuberculosis of the iris, the eye was enucleated. The microscope revealed a tubercular structure, but no bacilli were found. Hill Griffith justly pointed out that it was impossible to distinguish between primary and secondary tubercle of the iris. WERNER.

FERGUS (254) relates two cases of corectopia and one of small dislocated lens in one family. WERNER.

XIII.—CHOROID.

255. WOLKOWITSCH. Exenteration of the eyeball. *Mittheilungen aus klinischen und medicinischen Instituten der Schweiz.* From the eye clinic at Berne. Basle and Leipsic, 1894.

256. COLLINS, E. T. Case of a primary tumor of the ciliary body, of glandular structure. *Trans. Ophth. Soc. Un. K.*, xiv., p. 83.

WOLKOWITSCH (255) calls attention to the cases of fatal meningitis following enucleation, and suggests evisceration as the best means of avoiding this complication. 750 cases, 154 of them from the Berne clinic, have been reported without a fatal outcome. The indications for the operation in the Berne clinic are: 1, panophthalmitis of exogenous or endogenous nature; 2, the more plastic and less purulent forms of acute, subacute, and chronic irido-cyclitis that follow injury and generally lead to loss of function in the one eye and danger to the second. Recovery takes place in eleven days on an average. Neurotomy is only made in painful absolute glaucoma in Berne. He concludes with an accurate description of the technique and the antiseptic precautions observed.

COLLINS (256) refers to similar cases published by himself (vol. xi.), and by Alt, Michel, and Lagrange. In the present case, a woman aged twenty-eight, there was no evidence of disease in any other part of the body. The pupil was occluded from iritis. T+1. The tumor was situated in the upper part of the ciliary region, extending from the root of the iris to the equator. The anterior part was black, the posterior white. The growth presented a glandular appearance, with sarcomatous-looking collections of cells in the pigmented portion. Werner.

XIV.—GLAUCOMA.

257. Kugel. A case of return of qualitative perception of light after iridectomy in an eye amaurotic from glaucoma. *Graefe's Archiv*, xl., 3, p. 299.

258. Smith, Priestley. An instance of hereditary glaucoma, and its cause. *Ophth. Review*, xiii., p. 215.

In Kugel's (257) patient, blind for three weeks, a double iridectomy was done in each eye at an interval of two weeks, and the patient was able to count fingers in the right eye at four feet. The deep excavation disappeared, and the vessels which had emerged at the margin of the disc again appeared in the centre.

Priestley Smith (258) records cases of glaucoma in both eyes in a father and daughter, the latter aged twenty-nine. The eyes had been lost from absolute glaucoma with the exception of one of the daughter's eyes, in which vision was improved from motion of hand to $\frac{6}{24}$, by scleral puncture, followed by iridectomy. There was H = 6 D. The cornea measured 10.5 *mm* in the horizontal diameter in the father's eyes, and 10 *mm* in the daughter's eyes. The right eye of the daughter was excised for absolute glaucoma, and its dimensions were found to be subnormal, although the lens was slightly larger than normal. The filtration angle was closed. Small eyes generally contain disproportionately large lenses, due, according to the author, to the fact that the lens, in its capsule, has no vascular connection with the surrounding parts and is to a certain extent independent in its growth. Hereditary glaucoma would therefore seem to be caused by hereditary smallness of the eyeballs. Small eyes are attacked by glaucoma earlier in life than eyes of normal size; moreover, the recorded cases of hereditary glaucoma occurred at an early age.

Primary glaucoma seems to depend on various causes: sometimes on systemic, vascular, or nerve disturbance; sometimes chiefly on individual peculiarity in the build of the eye; and often on a complex co-operation of several causes. The author speaks favorably of scleral puncture as an adjunct to iridectomy, after several years of experience. Werner.

XV.—SYMPATHETIC OPHTHALMIA.

259. Rohmer. Resection of the optic nerve according to de Wecker in sympathic ophthalmia. *Ann. d'Ocul.*, cxii., p. 249.

260. Bronner. Case of sympathetic ophthalmia after concussion of the eyeball, with no visible external wound. *Trans. Ophth. Soc. Un. K.*, xiv., p. 215.

Rohmer (259) saw sympathetic inflammation follow resection of the optic nerve, making enucleation of the first eye necessary and permanently damaging the sight of the second. He favors enucleation solely in these cases.

Bronner's (260) patient developed sympathetic ophthalmia with K. punctata, eighteen days after a kick on the other eye. In the injured eye three was a small scleral staphyloma but no conjunctival wound, tension was never lowered, and inflammation was diminishing when the left eye became affected. Werner.

XVI.—VITREOUS.

261. Dimmer. A case of hemorrhage between the retina and the vitreous. *Beiträge zur Augenheilk.*, xv.

262. Benson. A case of "monocular asteroid hyalitis." *Trans. Ophth. Soc. Un. K.*, xiv., p. 101.

Dimmer (261) reports a case in which a large hemorrhage occurred between the retina and vitreous and partially covering the disc in a patient with albuminuric retinitis.

Benson's (262) patient, a gentleman, aged sixty-two, complained of slight asthenopia and muscæ. $V = \frac{6}{9}$ with each eye. Both eyes were normal with the exception of the vitreous humor in the right eye, which was studded with small, smooth spheres, like stars in the sky ; they did not sparkle like cholesterine crystals. They were fixed, and by oblique illumination looked cream colored. No change occurred during nine months' observation.

Werner.

XVII.—LENS.

263. Dalganoff. On the corneal astigmatism after extraction of cataract. *Wjest. Ophth.*, 1894, July-October.

264. Walkoff. Is capsulotomy necessary in the extraction of senile cataract? *Ibid.*

265. Wicherkiewicz. The after-treatment of cataract patients after infection has occurred. *Resport of the Society of Natural Scientists and Physicians*, Vienna, 1894.

266. Czermak. On pressure bandages and rupture of the wound after extraction. *Wiener klin. Wochenschr.*, No. 27, p. 506.

267. THOMPSON, L. L. Observations on some phases of opacity, and on luxation of the crystalline lens. *Ophth. Review*, xiii., p. 313.

268. SNELL. Case presenting unusual appearances after extraction of cataract; simulating a cyst, really due to a distended capsule. *Trans. Ophth. Soc. Un. K.*, xiv., p. 135.

WALKOFF (264) gives a detailed report of 303 cataract operations done on 181 patients between 1891 and 1893, according to his method, which is as follows. The section is made in the cornea, the highest point coming a little above the upper margin of the pupil. The lens with its capsule is expelled by simultaneous pressure above and below with two spatulas. No iridectomy is done. The advantages of this operation are a round pupil, high acuteness of vision and simplicity of execution. The bandage is not removed for a week or longer. Complications and unfavorable results are not more frequent than with other methods of operation. In 22 cases the cataract was immature but the results were good in all. In 48 cases the capsule ruptured, but in 36 of these it was removed with forceps. There was loss of vitreous in 96 cases, in one of which the lens was not extracted. In 2 cases there was severe hemorrhage leading to atrophy. Traumatic simple iritis and irido-cyclitis occurred in 102 cases, in 49 of which there was prolapse or incarceration of the iris. Severe irido-cyclitis took place in 16 cases, in 4 of which division of the membranes gave vision of $\frac{8}{200}$ to $\frac{10}{200}$. Purulent keratitis and panophthalmitis occurred in 8 cases; incarceration of the iris in 72. $V = 0$ in 16 cases, in 10 of which the blame rested on the patient. HIRSCHMANN.

In beginning infection after extraction, WICHERKIEWICZ (265) advises sterilizing the margins of the wound with the galvano-cautery, 4–10 % nitrate of silver, or bichloride. If there is already exudation in the anterior chamber, the wound should be opened and the chamber washed out with physiological salt solution. Against infection of the vitreous we are powerless. For the inflammation, mydriatics should first be used, with cold applications in the early stages, and warm ones later.

CZERMAK (266) believes that prolapse of iris is best prevented by making a large section in the sclero-corneal margin. A pressure bandage favors rupture of the wound. For this reason he has discarded the bandage, and uses Fuchs's double shield.

THOMPSON (267) describes: 1. Segmental opacities of the lower and inner portion of the lens. They are generally non-progressive, and occur in both eyes. The youngest patient was aged forty. 2. Annular opacities, or "arcus senilis lentis," the youngest patient being twenty-three. This form of cataract produces a spotted opacity of the anterior part of the lens after middle age. All the patients were women. 3. A case of fluid degeneration of cataract with restoration of vision. 4. Ectopia lentis. Four cases in relatives; all were highly myopic, with partial or complete iridodenesis, and the lenses were displaced outwards. Luxation of senile cataracts downwards is also referred to. WERNER.

In SNELL'S (268) case the lens was removed with vectis, with little loss of vitreous. $V = \frac{6}{9}$. Six or seven years later patient noticed a small white body in the ant. ch. There was a pear-shaped cyst at the lower part, attached to the iris by a slender filament, and was discovered to be the capsule of the lens, after removal. WERNER.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

269. POSSAUER. On the duration of life after the appearance of albuminuric retinitis. *Beiträge zur Augenheilk.*, xv.

270. DIMMER. The macular changes in retinitis albuminurica. *Prager med. Wochenschr.*, No. 42, 1894.

271. HUSEMANN. On tobacco amaurosis. *Deutsche med. Wochenschr.*, No. 43, 1894.

272. PETERS. On the occurrence and significance of the so-called displacement type of visual field. *Deutsche Zeitschr. f. Nervenheilk.*, v., p. 303.

273. KUNN. A case of embolism of a branch of the central artery of the retina, with remarks on the course of the macular arteries. *Wiener med. Wochenschr.*, No. 35, 1894.

274. LOMBARD. On a case of transient amblyopia following lead poisoning. *Rev. méd. de la Suisse Romande*, March 20, 1894.

275. WINTERSTEINER. On the structure, development, and genesis of glioma. *Wiener klin. Wochenschr.*, No. 27, p. 493.

276. BATTEN and SPICER. Congenital pigmentary plaques of retina. *Trans. Ophth. Soc. Un. K.*, xiv., p. 127.

277. BRAILEY. Notes of a case of failure of central vision. *Ibid.*, xiv., p. 123.

278. SPICER. Case of retinitis circinata. *Ibid.*, xiv., p. 132.

279. Griffith. On a rare form of primary intra-ocular melanoma. *Ibid.*, xiv., p. 160.

280. Collins, E. T. Two cases, brother and sister, with peculiar vascular new growth, probably primarily retinal, affecting both eyes. *Ibid.*, xiv., p. 141.

The investigations of the Baroness Possauer (269) include 131 cases of albuminuric retinitis in chronic nephritis. It is not known exactly how often retinitis occurs in Bright's disease, the figures varying between 6 and 33 %. Many patients die soon after the appearance of the retinitis, but there were 5 cases in which the albuminuric retinitis was cured permanently. Among the Zurich patients the mortality of the men was greater than that of the women. Five women were living more than two years after the first ophthalmoscopic examination, and one eleven years after.

In eyes which showed ophthalmoscopically the macular star figure of retinitis albuminurica, Dimmer (270) found only collections of fatty degenerated cells in the outer nuclear layer of the retina. The stellate arrangement of the patches is due to the radiating arrangement of the cone fibres in the outer nuclear layer. Herrnheiser.

Husemann (271) gives a short report on epizoötic blindness in horses in Australia. The eyes are not inflamed, as in the forms previously known, but the disease leads to complete blindness in a year or two if the animal is not removed from the district. The disease is due to the Australian tobacco, *Nicotiana suaveolens*, and consists in an atrophy of the optic nerves.

Peters (272) gives a *résumé* of previous articles on the displacement type of the limits of the visual field, and reports the findings in 150 patients examined by himself. In 98 cases it was wanting. Of the 52 cases showing it, 19 had nervous disturbances, while the rest were injured but had no nervous affection. In traumatic neurosis the symptom has no significance, being found in about half the cases. In 104 healthy individuals, the type was found in 23 % of cases.

In Kunn's (273) case there was an embolism of the superior temporal branch of the central artery of the retina in a student, aged sixteen, with mitral insufficiency. There was a scotoma and normal central vision, due to the fact that the macula is mostly supplied by the small macular arteries which are sometimes given off low down, and which may then continue to supply the macula even when the main artery is blocked above them.

Lombard's (274) patient was a young man with lead-poisoning, who had an attack of colic followed by complete blindness. The pupillary reflex was lost and the discs were pale. The following day he counted fingers at 12 feet, and two days later the vision was so far restored that he could go out. Sulzer.

Vision (276) was normal. No abnormality of fundus, except the plaques which were three or four in number, two being large; they are almond-shaped, with the pointed end directed towards the disc. They are red-gray in color, and not raised or depressed. The red reflex appears to shine through them, and the retinal vessels pass over them. Werner.

Brailey's (277) patient, a man, aged fifty-seven, had slight pigmentary changes at the macula, with an area of superficial choroidal atrophy around the disc and m. l. Some of the choroidal vessels had thickened coats. Werner.

Spicer's (278) patient, a woman, aged seventy-three, presented in the left eye the typical appearance of retinitis circinata, as described by Fuchs, without any trace of hemorrhage. In the other eye there were numerous hemorrhages without any white spots. Werner.

In Griffith's (279) case the melanotic growth sprang from the retinal pigment epithelium near the ciliary region; it was composed of columns and tubules of epithelial cells. After enucleation the growth recurred in the orbit and superior maxilla, and the patient, a man, aged thirty-seven, died within a year.

Werner.

In Collins's (280) first case the man's sight began to fail at age of twenty. There was detachment of the retina with enormous dilatation of vessels and development of white dots on its surface. Subsequently there was increased vascularity of the iris with glaucoma. The eye was enucleated, and the retina showed extensive changes, containing in one part a close plexus of small vessels. The sister became blind at twelve years of age, both eyes were staphylomatous, cornea opaque, and a dark bulging existed at the outer canthus of one eye. Microscopically both eyes contained bony plates in the choroid, no lens, iris, or ciliary body visible. A large mass involving the cornea in one eye existed in the globes, composed of small branching capillary vessels, having between them honeycombed cells with large nuclei.

Werner.

XIX.—OPTIC NERVE.

281. Elschnig. On choked disc. *Report of the Society of German Natural Scientists and Physicians in Vienna*, 1894.

282. Moll. A case of recurrent retrobulbar neuritis. *Centralbl. f. prakt. Augenheilk.*, xviii., p. 269.

283. Juler. Uniocular optic neuritis. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 121.

284. Sandford. Case of double optic neuritis from caries of sphenoidal cells and intracranial abscess. *Ibid.*, p. 119.

285. Williams, R. A case of double optic neuritis. *Ibid.*, p. 118.

286. Taylor. Optic neuritis in its relation to intracranial tumor and trephining. *Ibid.*, p. 105.

Elschnig (281) calls those cases choked disc only when the disc projects 2 D. The examination of 55 cases showed an inflammation of the disc in so far as the discovery of increase in nuclei and infiltration of the central connective-tissue tract. The swelling is due to œdema, which never affects the lamina cribrosa. The optic nerve always shows changes in the form of interstitial neuritis and perineuritis. Hydrops of the optic-nerve sheath was not constant. In 21 cases of brain tumor inflammation in the meninges was found 13 times.

Moll's (282) case was that of a young man in whom a retrobulbar retinitis developed in both eyes, and recurred three times in four years. The function remained good, while the discs showed white atrophy. The cause was probably a chronic periostitis about the optic foramen.

Juler's (283) patient, a woman aged twenty-eight, had syphilis; hemicrania was the only other symptom beside the loss of vision. No P L. Werner.

The only observable symptoms in Sandford's (284) case, a man aged seventy-eight, were blindness and optic neuritis two years and a half before death, which took place without paralysis or previous loss of consciousness. There was a large encysted abscess at the base, between the layers of dura mater in the middle fossa. The walls of the sphenoidal sinus were completely destroyed. Werner.

Williams's (285) patient, a woman aged sixty-five, suddenly lost her sight, without any symptoms, headache or otherwise. There was slight haziness of the discs, pupils dilated and inactive.

She was given potass. iodid., and in six weeks V = $\frac{6}{18}$. Fields normal. Neuritis gone. WERNER.

TAYLOR (286) records nine cases of trephining for cerebral tumor, in which optic neuritis was present. In the first three cases the tumor was removed and the optic neuritis subsided; in one of these the discs were normal four months after operation, and in another the tumor recurred, but only slight optic neuritis reappeared in the final stage. In the second group twice the tumors were seen, but not removed, and in a third no growth could be found, nevertheless the neuritis subsided, although in one case the tumor continued to grow. In the third group, the tumors were removed, but recurred in two cases without redevelopment of neuritis. A cyst was tapped in the last case, with subsidence of the neuritis, although the patient died of tumor. No active treatment by drugs was carried out after operation. These cases seem to favor the view that increased ntracranial pressure is the effective agent in producing optic neuritis. In the discussion, Mr. V. Horsley stated that there were a sufficient number of cases of recurrence of the growth without recurrence of the neuritis to show that even if the dura mater be not opened, the optic neuritis disappears. WERNER.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

287. SCHIRMER. The Mackeown-Hirschberg method of magnet extraction. (An answer to Prof. Hirschberg.) *Deutsche med. Wochenschr.*, No. 29, 1894.

288. PERLES. On the removal of foreign bodies from the eye. *Berl. klin. Wochenschr.*, No. 28, 1894.

289. HIRSCHBERG. On the removal of iron splinters from the retina. *Deutsche med. Wochenschr.*, Nos. 25 and 26, 1894.

290. ARGYLL-ROBERTSON. Case of trichosis bulbi. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 196.

Since Haab's publication, many objections have been made to HIRSCHBERG'S (289) method of extracting foreign bodies from the eye, and some have thought that the results were not good. Hirschberg has done the operation 150 times, and has never seen suppuration in the wound, and suppuration in the interior but once. The careful introduction of a clean magnet is not dangerous. Loss of vitreous is rare, and bruising of the vitreous is avoided when a sharp knife is first passed through the vitreous in the direction of the foreign body. Foreign bodies whose location is unknown may be removed with a small magnet. The bad results depend chiefly upon the severity of the injury. Sometimes the foreign bodies are so firmly imbedded that removal is impossible.

This (290) was a dermoid situated on the upper and outer part of the sclerotic under the upper lid; a few large hairs grew from its surface. WERNER.

XXI.—OCULAR DISTURBANCES IN GENERAL AFFECTIONS.

291. WESTHOFF. Diabetic inflammations of the eye. *Medisch. Weekbl.*, p. 416, 1894.

292. PEL. Hemianopsia. *Ibid.*, p. 407.

293. ANDOGSKY. On eye inflammations from intestinal worms. *Zehender's klin. Monatsbl.*, xxxii., p. 263.

294. BERGMEISTER. On ocular disturbances with certain anomalies of metabolism, particularly the increase of uric acid. *Report of the Society of Natural Scientists and Physicians in Vienna*, 1894.

295. BOGMANN. Contributions to the study of ocular affections during lactation. *Ann. d'Ocul.*, cxii., p. 161.

296. FREYHAN. A case of tubercular meninigitis that recovered. *Deutsche med. Wochenschr.*, No. 36, p. 707.

297. KNAPP. A case of otitic brain abscess successfully operated. *Arch. of Otol.*, xxiv., p. 155.

298. AXENFELD. On purulent metastatic ophthalmia, its etiology and prognosis. *Grafe's Archiv*, xl., 3 and 4.

299. BENSON. A case of recurrent transitory blindness. Eighth International Congress. *Arch. f. Augenh.*, xxix., 3–4, p. 378.

300. DEJERINE and VIALET. On a special form of functional hemianopsia in neurasthenia and traumatic neurosis. *Ann. d'Ocul.*, cxii., p. 147.

301. STERN. On periodic variations in the functions of the cerebral cortex. *Berl. klin. Wochenschr.*, 1894, p. iiii.

302. SEREVRENNIKOWA. A case of amaurosis due to gumma of the base of the brain. *Wjest. Ophth.*, 1894, June–October.

303. IWASA. On a case of tubercle in the optic thalamus. *Inaug. Dissert.*, Freiburg, 1894.

304. Wernicke. Complete right homonymous hemiopia after a perforation wound in the left occipital region. *Deutsche militärärztliche Zeitschr.*, 1894, part ii.

305. Jacob. On a case of hemiplegia and hemianæsthesia and crossed oculomotor paralysis with unilateral destruction of the optic thalamus, the posterior part of the internal capsule, and the anterior corpus quadrigeminum, with particular reference to the secondary degenerations. *Deutsche Zeitschr. f. Nervenheilk.*, x., pt. 2 and 3.

306. Wilbrand and Saenger. On the visual disturbances in functional nervous affections. Leipsic, 1892.

307. Guibert. Poliencephalitis superior and inferior. Recovery. *Arch. d'opht.*, xiv., 9, p. 543.

308. Long and Beevor. Binasal hemiopia in a case of tabes dorsalis. *Trans. Ophth. Soc. U. K.*, xiv., p. 246.

Westhoff (291) describes a case similar to Horner's, in which the refraction changed in a diabetic patient. A man of fifty-one stated that four days previously he suddenly found that he saw poorly near by and clearly in the distance. Previously he had worn — 1.D for distance and read without glasses. The patient was found to have hyperopia in one eye and hyperopic astigmatism in the other. Fourteen days later the patient had myopia again and read without glasses.

Pel (292) demonstrated a patient aged forty-two, who suddenly noticed that he could not see the right half of his paper. There was also disturbance of intellect and speech, and paralysis of the right leg and face. The hemianopia was permanent, the other symptoms dissappeared. Westhoff.

According to Andogsky (293) a great number of the eye diseases observed in children with intestinal worms depend upon the disturbances in the formation and circulation of the blood thus caused. The disturbances are usually functional only, but there may be neuro-retinitis and hemorrhages in the retina. Reflexly there may occur mydriasis, myosis, spasm of accommodation, strabismus, and blepharospasm. Andogsky describes a unilateral blepharospasm in a girl of fifteen and a bilateral blepharospasm in a girl of eleven, which resisted every form of treatment, but disappeared at once after the removal of the worms.

In 4 cases with increase of uric acid in the urine, Bergmeister (294) found posterior polar opacities of the lens, which disappeared as the uric acid grew less. He also found retinal hemorrhages and neuro-retinitis.

Bogmann (295) sketches the history of visual disturbances during lactation from their earliest description to the present. His own patient had a double optic neuritis coming on in the third week after parturition following the sudden cessation of the secretion of milk. Sulzer.

Freyhan's (296) patient, a man of twenty, had the usual clinical symptoms of tuberculous meningitis, the diagnosis being confirmed by the discovery of tubercle bacilli in fluid obtained by puncture. Ophthalmoscopically there was neuro-retinitis. The case recovered completely.

In a girl aged nine, Knapp (297) found chronic otorrhœa, headache, fever, choked disc, and homonymous hemiopia. On account of the general symptoms and the absence of other nerve affections, an otitic cerebral abscess was diagnosed and a radical mastoid operation and trephining were done. An abscess in the temporal lobe was evacuated. A cerebral hernia resulted. The general condition was bad until two small superficial abscesses were evacuated, when the hernia retracted and the child recovered.

Axenfeld (298), for the purpose of determining the prognostic significance of metastatic ophthalmia, reports 14 cases of his own examined clinically and bacteriologically, and reviews the 200 cases in the literature.

His conclusions are as follows: Purulent metastatic ophthalmia is complicated with ulcerative endocarditis in one third of the cases. To this, with the frequent degeneration of thrombi is due the frequency of the diseases after parturition. In one third of the cases it is bilateral and when following parturition is almost always fatal. Unilateral metastatic ophthalmia often accompanies slight attacks of pyæmia, particularly the cases without assignable cause, and those following affections of the lungs or meninges. It is mostly an affection of the capillary vessels of the retina, as is shown by the fact that, although blindness comes on rapidly, it never occurs suddenly, as it would if larger vessels were stopped. Often the ocular metastasis is the only one found.

In the puerperal processes the cause is the streptococcus in most cases, in surgical cases this and the staphylococci, these micro-organisms usually causing panophthalmitis. The Fränkel-Weichselbaum pneumococcus is the cause of some cases and these usually end in phthisis without panophthalmitis.

According to the observations of Dejerine and Vialet (300) concentric contraction of the visual field is not the only form of limitation of the field found in neurasthenia and traumatic neurosis. Both affections can cause hemiopia, which, in spite of long duration, is only functional and has no anatomical foundation. It differs from ordinary hemiopia in the marked variations of the limits of the preserved half of the field. In two patients, one with extreme spontaneous neurasthemia and one with traumatic hysteria, the authors had observed the type of hemiopia described, during four years. SULZER.

The new variety of nervous functional disturbance described by STERN (301) consists in a periodic decrease in the function of the cerebral cortex. There is an intermittent decrease in the sensibility of all the sensory regions, a paresis with ataxy of the voluntary muscles, and a decrease in intellectual power. In one case the vision, usually $\frac{2}{3}$, decreased to $\frac{1}{12}$, with simultaneous contraction of the field.

SEREVRENNIKOWA'S (302) patient, a man of twenty, five months after the chancre for which he had received mixed treatment, developed severe headache, which, in spite of treatment, was soon followed by loss of sight in the right eye. In two weeks the left eye was completely blind and the right had begun to fail, and was blind ten days later. Following this there was paralysis of both oculomotor nerves. Ophthalmoscopically nothing abnormal was found. The sense of smell was weakened, and the forehead and scalp were sensitive to pressure. A diagnosis of gumma behind the chiasm was made. At the autopsy, eight months after infection, there was found a gumma as large as a walnut in the cerebral substance, behind the chiasm, above the left optic tract, and a smaller gumma more to the right. HIRSCHMANN.

WERNICKE'S (304) case was peculiar from the fact that the hemiopia was the only symptom of the cerebral injury. The first occipital convolution must have been affected. The transitory ptosis was thought to be due to the influence exerted from a distance on the gyrus angularis which lies anterior and is probably the cortical centre for the extrinsic ocular muscles.

GUIBERT'S (307) case was a woman of twenty-five in whom within a short time severe headache developed and a paralysis, apparently bulbar, of all the ocular muscles on each side. Vision was reduced to $\frac{1}{4}$, field normal, and fundus normal. The urine contained sugar. Although the case did not appear to be syphilitic, it must be considered such, as it was completely cured by the injection of oleum hydrarg. biniodid. and the internal use of potas. iodid. followed by galvanization.

In this case (308) there was atrophy of discs. V $\frac{6}{24}$ and $\frac{5}{60}$. Red-green blindness. WERNER.

MISCELLANEOUS NOTES.

APPOINTMENTS.

Prof. I. SCHNABEL began his functions as director of the Second Eye Clinic in Vienna, April 3, 1895.

Prof. W. CZERMAK, of Innsbruck, has been appointed director of the Eye Clinic in the German Medical Faculty of Prague.

MEETINGS OF SOCIETIES.

BRITISH MEDICAL ASSOCIATION, in London, July 30th to August 2d, inclusive.

GERMAN OPHTHALMOLOGICAL SOCIETY, 4th to 7th August, at Heidelberg.

At the last meeting of the GEORGIA STATE MEDICAL ASSOCIATION an OPHTHALMOLOGICAL SECTION was organized. Dr. J. L. Hiers, of Savannah, was elected President, and W. F. Aiken, of Savannah, Secretary. Other members enrolled were: Calhoun, Hobbs, and Ray, of Atlanta; Hull and Tinsley, of Augusta; Shorter, of Macon; Paige, of Savannah.

Contents of the last number of the Archiv fur Augenheilkunde (German edition of these ARCHIVES).

Bd. XXX., Nos. 3 and 4, issued April, 1895.

A.—ORIGINAL PAPERS.

VIII. LAQUEUR. Embolism of the Central Retinal Artery with Integrity of the Temporal Portion of the Retina. Plate IV.

IX. C W. CUTLER. Congenital Night Blindness and Pigmentary Degeneration. Plates I. and II. (To appear in our next number.)

X. THE SAME. Three unusual Cases of Retino-Choroidal Degeneration. Plate III. (To appear in our next number.)

XI. L. CASPAR. Retinal Striæ. Plates V. and VI.

XII. W. SCHÖN. The Functional Diseases of the Ora Serrata and the Ciliary Part of the Retina.

XIII. N. ANDOGSKY. Operative Treatment of Trachomatous Xerophthalmus.

XIV. THE SAME. Formaldehyd, as a Preservative of Human Eyes for Operative Courses.

XV. E. GOTTBERG. Shot Blind in an Attempt at Suicide.

XVI. S. HOLTH. Light, our Yellow Ointment, and the Salve-Jars in common use.

XVII. THE SAME. Syphilitic Auto-infection and Hard Chancre of the Lids.

XVIII. L. BACH. Experiments on the Danger of Infection from Penetrating Injuries of the Globe when the Conjunctiva is Infected.

XIX. A. DARIER. Subconjunctival Injections. Reply to Drs. Mellinger and Gutmann.

XX. E. ADAMÜK. Two Cases of Malignant Glaucoma.

XXI. S. LACHOWITZ. The Bacteria of the Healthy Human Conjunctiva.

XXII. C. SCHWEIGGER. Note on the Mechanism of Accommodation.

B.—REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD QUARTER OF THE YEAR 1894. (Translated in this number.)

VOL. XXIV. No. 3

ARCHIVES OF OPHTHALMOLOGY.

OBSERVATIONS ON THE MACULA LUTEA.

By G. LINDSAY JOHNSON, M.A., M.D., CANTAB., F.R.C.S., London.

(*With 10 microphotographs in the text.*)

HISTOLOGY OF THE HUMAN MACULA.[1]

IN a former article on this subject (vol. xxi., pp. 1–21), I dealt with the ophthalmoscopic appearances of the healthy macula. I therein drew attention to the shape of the macula reflex, and to the internal reflex of the fovea, and expressed the belief that these reflexes, as well as those observable along the retinal vessels, were probably due to the inequalities of the surface of the tissues, which, if decreasing with age, would account for the gradual disappearance of these reflexes.

I thought that the only satisfactory way of testing this would be by microscopical examination of perfectly healthy retinæ of individuals of various ages.

At the very outset I was struck by the considerable differences which the retina presented in some of the most important points as compared with the descriptions generally given; and as I advanced it became more and more evident that, in order to understand the histology of the macula, I should have to re-examine the minute anatomy of the entire retina. I need hardly point out how difficult it is to obtain the necessary material for such research, as the retina is so highly differentiated, and therefore degenerates so rapidly, that the tissues of eyes require not only to be absolutely healthy, but they must be fixed with extreme rapidity. I should certainly not have succeeded but for the great assistance I received from Mr. Lawson, Mr. Power, Mr. Higgins, Mr. MacKinlay, Mr. Brailey, and Dr. Landolt, of Paris, whereby I was enabled to obtain a number of healthy eyes.

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[1] My friend Mr. Henry de Mosenthal has rendered me invaluable assistance in preparing this series of papers. He has examined my entire series of microscopic specimens, and I have had the advantage of discussing every point with him.

The methods of fixing, preserving, and preparing eyes as best suited for this particular work would, if here detailed, overburden this article. I therefore propose to discuss these methods on a future occasion, but wish to take this opportunity of thanking Mr. Bolles Lee and Dr. E. C. Bousfield for their kind advice and assistance.

I have made serial sections of eyes of individuals of different ages, ranging from the five-months fœtus to old age, and I propose in this and the subsequent papers to record my results, discussing the histology of the various layers of the retina in their order from without.

I have tried to illustrate my results by microphotographs, and hope that the accompanying process reproductions of them will be found more satisfactory than the ordinary drawings, which invariably leave much to be desired on the side of accuracy. Microphotographs, on the other hand, show such superabundance of detail that the salient points are less striking; and, as it is only possible to photograph in one plane, all parts cannot be in focus—defects which, to my mind, are more than compensated for by the elimination of the personal factor.

A.—THE HEXAGONAL PIGMENT LAYER.

From the study of the development of the eye, the hexagonal pigment layer is universally conceded to belong to the retina, but it is held to be histologically united to the membrana vitrea.

I find that the membrana vitrea is exclusively choroidal, and forms so intimate a part of the choroid that it cannot be detached from it without tearing and without the destruction of the vascular walls of the chorio-capillaris. I further find that between the membrana vitrea and the hexagonal pigment layer there is a space, the outer boundary of which is the membrana vitrea, and the inner also a distinct membrane, which I propose calling the "MEMBRANA TERMINANS RETINÆ."

The membrana terminans is connected with the membrana vitrea by very minute fibrillæ, similar to the retiform fibrillæ which we find connecting the surfaces of minute lymphatic spaces in many other parts of the body. Often I find the membrana terminans so near the membrana vitrea that the space between the two is not perceptible; then again I find the space between the two membranes very noticeable, the fibrillæ being stretched from the one membrane to the other almost at right angles, and between these two extremes every intermediate stage seems to exist. The following illustration shows the space between the membrana vitrea and the membrana terminans and the fibrillæ holding the two membranes together.

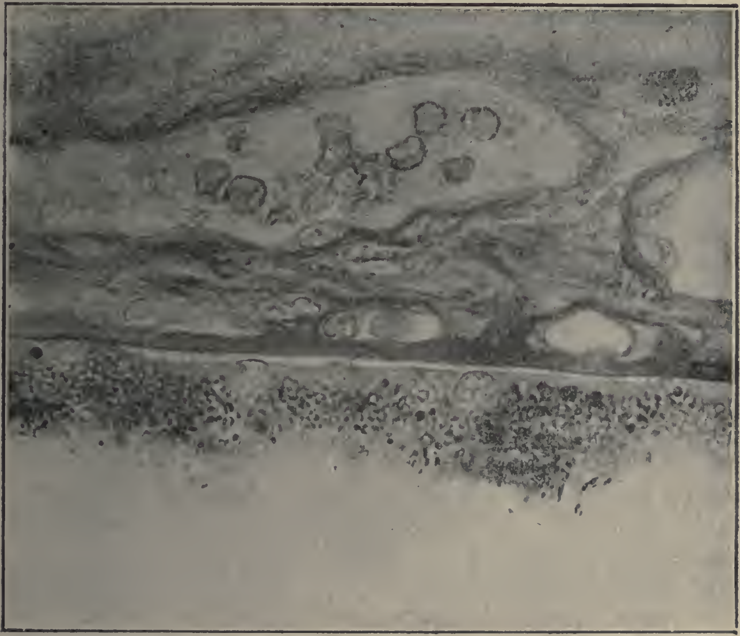

FIG. 1.[1]

Vertical section through the choroid and hexagonal pigment layer showing the space between the M. vitrea and M. terminans and the fibrillæ connecting them. Ehrlich-Biondi and nigrosin stain. x 1060 diams. (Zeiss 2 *mm* apo. obj.; 2 proj. ocular; camera extension 98 *cm.*)

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[1] All the microphotographs have been taken on half plates (6½" x 4¾") and then cut down to allow of printing with the text.

N.B.—*All the illustrations of vertical sections are placed relatively in the same position viz., with the external portion upwards.*

In many of my specimens I find the hexagonal pigment layer detached from the choroid, and in those cases I can, as a general rule, see the torn ends of the fibrillæ adhering partly to the membrana vitrea, and partly to the membrana terminans.

As to the hexagonal pigment layer itself, it is permeated to such an extent with pigment that it is excessively difficult to distinguish its structure clearly unless very thin sections be made.

It seems to me that what have until now been taken for the nuclei of hexagonal cells are independent spherical corpuscles (spherules), as shown in the following figure.

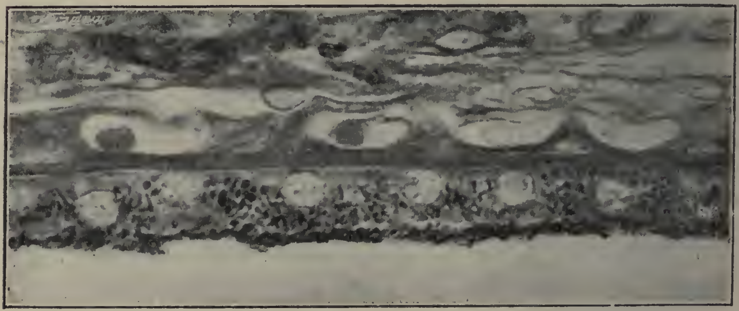

FIG. 2.

Vertical section through the choroid and the hexagonal pigment layer showing round spherules resting against the membrana terminans. Ehrlich Biondi and nigrosin stain. x 1180 diam.[1] (Zeiss 3 *mm* apo. obj. No. 4 proj. ocular; camera extension 90 *cm.*)

These spherules are all of the same size and equidistant; they lie much closer together in the macula region than in any other part of the retina, and nearly touch each other at the fovea centralis, being farther apart the nearer the ora serrata. This seems to indicate that the spherules play an important part in vision.

In eyes which have been subject to pressure, as is the case in glaucoma and certain ocular tumors, I find the spherules appearing horizontally oval, in section, just as a rubber ball would appear if compressed by a weight; and this seems to point to the spherules being elastic. Artificial pressure during the preparation of the specimen will also sometimes produce the same effect.

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[1] In order to ensure accuracy the magnification of the illustrations have in every case been determined with the micrometer scale placed on the stage and focussed on to the photographic screen.

The following photograph which illustrates the above is taken from a section of an eye removed for persistent glaucoma with high tension.

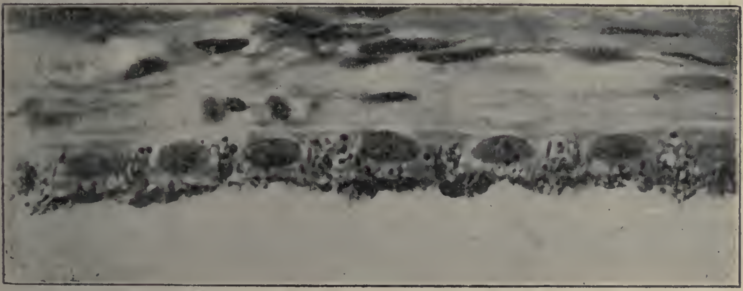


FIG. 3.

Vertical section through choroid and hexagonal pigment layer, showing oval spherules. Hæmatox. stain. x 1180 diam. (Zeiss 3 *mm* apo. obj.; No. 4 proj. ocular; camera extension 90 *cm.*)

In some specimens I have noticed the spherules entirely detached, and frequently distinct spaces are observable around the spherules, which I ascribe to shrinkage during the dehydration of the specimen.

I need hardly say that I have had considerable hesitation before calling into doubt the existence of nucleated hexagonal cells in this layer. I examined the eye of an albino rabbit, the section of which showed the spherules quite clearly, and I have seen the same appearance in sections of the eyes of various animals.

The fact that in some cases these spherules are entirely detached and float free in the field of the microscope, the fact that I have never seen hexagonal cells isolated or even partially detached from each other along the line of the septa, and lastly the fact that spaces can frequently be seen around these spherules when in situ, all point to their not being nuclei. Moreover, the regularity of the spherules and their general appearance afford me further grounds for believing that the hexagonal pigment layer has a different structure to that usually accepted.

I would describe it as formed of two entirely different superposed layers.

1. THE OUTER LAYER—THE GELATINOUS LAYER,—which is the true hexagonal pigment layer, consists of a gelatinous homogeneous stratum, which stains uniformly throughout, limited externally by the membrana terminans retinæ and having internally a well defined boundary parallel to it. In this gelatinous layer spherules lie embedded at equal distances. Around each spherule, spherical pigment granules are embedded in the gelatinous matrix, like currants in a jelly. These granules become more numerous the further they are from the spherules, and form distinct lines of polygonal shape, being separated from the granules which surround the spherule in the next area by a clear line of the gelatinous matrix. The granules which are embedded on each side of this clear portion are arranged in a straight line. Viewed vertically no separations are visible, and the pigment granules cease wholly in the vicinity of the membrana terminans so as to leave a clear portion at the outer edge of the layer.

If the usual description of hexagonal cells held together by cement were correct, septa ought to be perceptible in vertical sections. This, however, is not the case. Pigmented streaks are perceptible where the pigment granules lie closest, and these streaks become more marked when the gelatinous layer has been subjected to pressure.

Transverse sections, when stained with Biondi-nigrosin, show the gelatinous layer as a homogeneous pale blue matrix containing the spherules and permeated with pigment granules. As the granules are absent in the immediate vicinity of the membrana terminans, transverse sections, when very slightly oblique, show at the edge the clear hyaline layer containing spherules but free from pigment granules. In these clear portions no trace of septa can be seen, as shown in the following photograph.

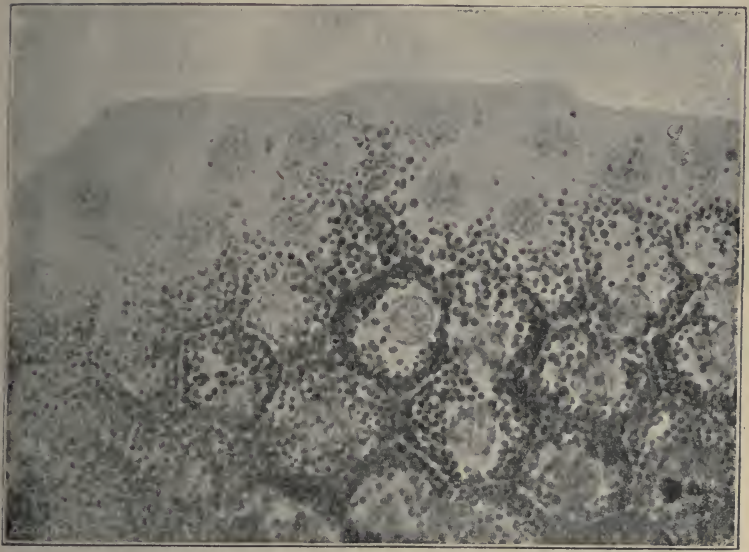


FIG. 4.

Transverse section (slightly oblique) through the hexagonal layer showing hexagons and at the border the clear hyaline layer interspersed with spherules. x 1080 diam. (Zeiss 2 *mm* apo. obj.; No. 2 proj. ocular; camera extension 100 *cm.*)

It seems to me hardly likely, if the septa exist, that the cement and cells would stain so uniformly as to be indistinguishable; I have not been able to differentiate them with any of the known stains or lakes. What have been taken for distinct septa therefore seem to be nothing more than clear portions of the gelatinous stroma lying between two parallel lines of pigment granules.

2. THE INNER LAYER—THE PIGMENT CRYSTAL LAYER,—consists of ill-defined irregularly strewn clusters of acicular or oat-shaped pigment crystals, entirely distinct in shape and appearance from the pigment granules which are embedded in the gelatinous layer.

The following figure shows the granules and the crystals. The boundary line of demarcation between these two distinct forms of pigment shows less clearly in this very highly magnified illustration than in Fig. 2.

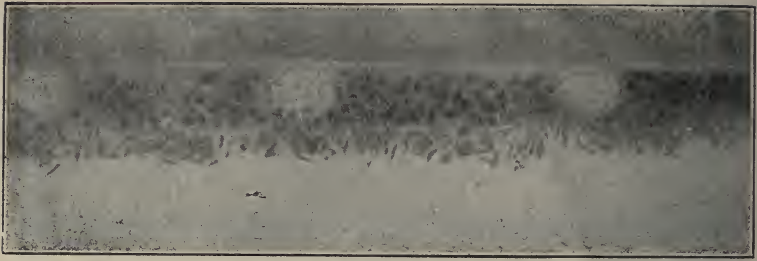


FIG. 5.

Vertical sections through the hexagonal pigment layer showing the two forms of pigment. x 1560 diam. (Zeiss 2 *mm* apo. obj.; No. 2 proj. ocular; camera extension 145 *cm.*)

Whilst the round pigment granules are firmly embedded in their gelatinous matrix, the acicular pigment crystals seem free to move. They lie in clusters in and adhere to a network—which I propose to term THE PIGMENT CRYSTAL PLEXUS—consisting of excessively fine fibre-threads lying like a thin gauze, two, three, and sometimes four meshes deep, on the gelatinous layer to which it is organically united.

In undisturbed specimens of vertical sections the plexus is usually invisible, being entirely obscured by the dense clusters of crystals. In order to show this plexus I have therefore had to select a specimen in which the pigment crystal layer has been disturbed and the plexus become partly uncovered. The following figure represents such a specimen:

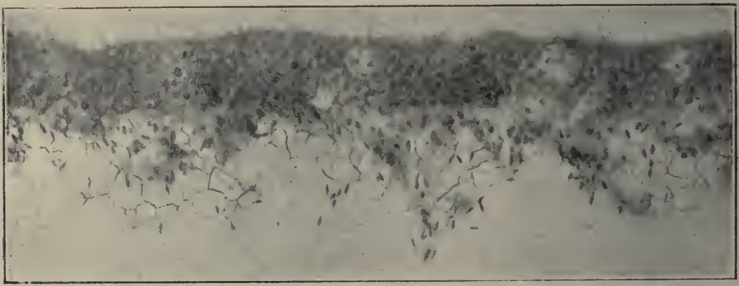


FIG. 6.

Vertical section of hexagonal pigment layer with disturbed pigment crystal layer showing the pigment crystal plexus. Hæmatoxylin stain. x 1000 diam. (Zeiss 3 *mm* apo. obj.; No. 2 proj. ocular; camera extension 134 *cm.*)

The acicular crystals are so highly refractive as to make it very difficult to define their borders, but they always appear elongated. I have never found them outside this plexus, nor in the gelatinous layer, except in specimens which have been subjected to pressure or disturbed in manipulation.

I have not been able to trace exactly how the crystal pigment plexus is connected with the gelatinous layer and the spherules. That, however, they are organically united seems to be clearly demonstrated by the fact that, in specimens which show the retina detached from the choroid by a rupture of the fibrillæ between the membrana vitrea and the membrana terminans, the whole hexagonal pigment layer is drawn away by the bacillary layer of which the crystal pigment plexus forms a part.

Transverse sections through the crystal pigment plexus show that the meshes are of the same shape and size as the hexagons of the gelatinous layer.

To illustrate this Figs. 7 and 8, of equal magnification, are here given.

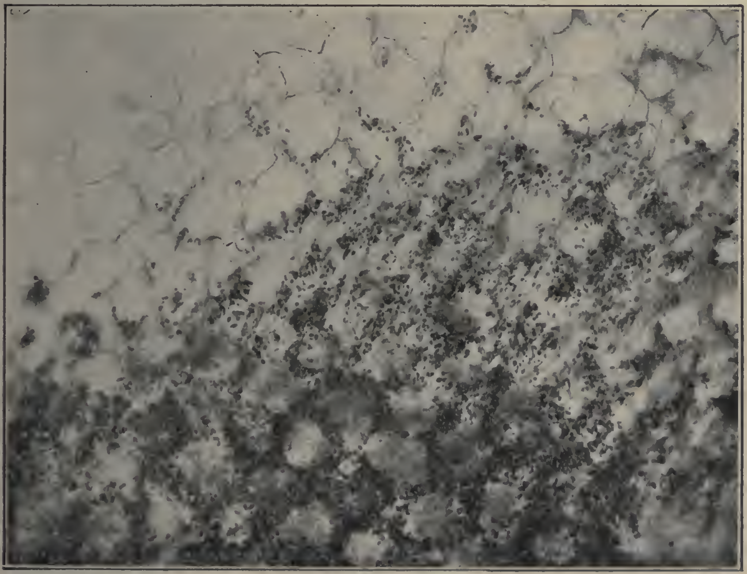


FIG. 7.

Transverse section through the crystal pigment plexus. x 560 diam. (Zeiss 3 *mm* apo. obj. ; No. 2 proj. ocular ; camera extension 83 *cm.*)

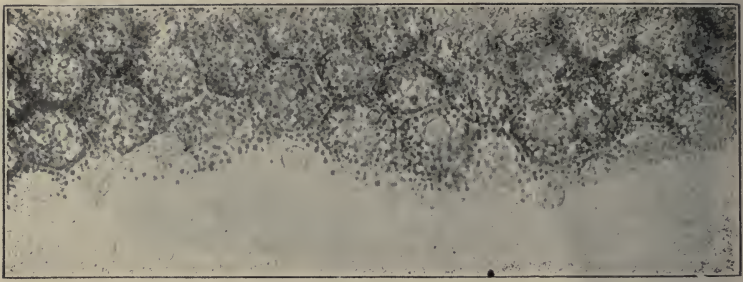


FIG. 8.

Transverse section through the gelatinous layer. x 560 diam. (obj., etc., as in Fig. 7).

A comparison of the two last figures leads to the assumption that the crystal pigment plexus penetrates the gelatinous layer along the clear spaces left between the pigment granules as far as the hexagons extend. It seems likely that there this plexus is connected with the spherules by fine fibrillæ, as in specimens in which the gelatinous layer has been disturbed I have been able to trace fine fibrillæ adhering to the spherules. The following figure shows such a specimen.

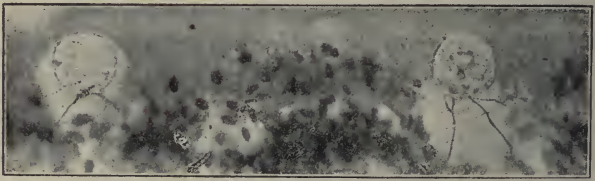


FIG. 9.

Vertical section through the disturbed gelatinous layer showing spherules with fibrillæ attached. x 1620 diam. (Zeiss 2 *mm* apo. obj.; No. 2 proj. ocular; camera extension 142 *cm.*)

We shall, when discussing the bacillary layer, find that the crystal pigment plexus is inseparably bound to it, so that the retinal nerve fibrils most probably terminate inside these spherules.

Owing to the minuteness of the spherules and the difficulty of obtaining differential staining it is not easy to analyze their internal structure. It seems, however, as if the nerve fibril or fibrils terminate in a bulbous end in the centre of the spherule, there kept in position by a number of fine radiating fibres embedded in a granular substance which fills the spherule. Indications of this will be found in Figs. 2, 5, and 9, but I have specially photographed a spherule with very high magnification. I thought it well, in this instance, to add to the photograph of a human spherule that of a spherule from the eye of a monkey.

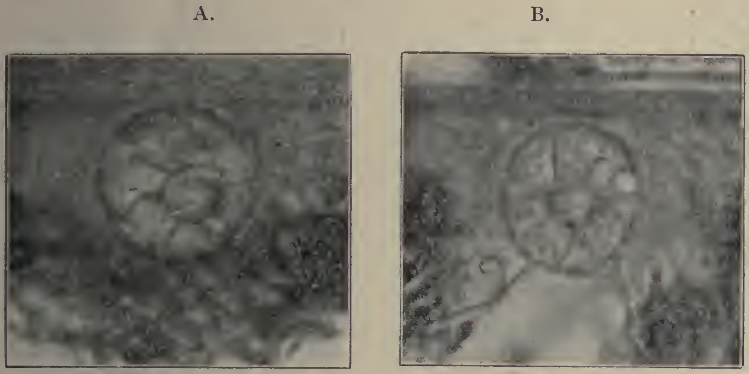


FIG. 10.

Vertical section of spherules. x 3000 diam. (Zeiss 2 *mm* apochr. obj.; No. 4 proj. ocular; camera extension 142 *cm*.)

(A) From a man.
(B) From a monkey *(Cercocebus Fuliginosus)*.

To recapitulate, then—the membrana vitrea is the limit of the choroid—then follows a space—then the membrana terminans retinæ—then a gelatinous layer in which spherules lie surrounded by embedded spherical pigment granules—lastly, the acicular pigment crystals lying in irregular clusters in a plexus.

This plexus is a direct continuation of the bacillary layer, and enters the gelatinous layer to terminate at the spherules, which latter seem to be closely connected with, and may essentially be found to contain, the ultimate terminals of the optic nerve fibres. At the macula the spherules lie much closer together than in any other part of the retina and nearly touch each other at the fovea centralis.

Without, at this stage, entering into the discussion of any of the physiological and other questions which the above description raises, I may briefly state that I am led to account for the most frequent form of detached retina as being due to the tearing of the bacillary layer, whilst the less serious and rarer form is due to the tearing of the fibrillæ which hold the hexagonal layer to the choroid.

The tearing of the bacillary layer is of course a much graver accident as regards vision than when the hexagonal layer remains in uninterrupted continuity with the rest of the retina. In this latter case the detached retina is still capable of some vision, since the retinal arteries supply to some extent the functions of the choroidal vessels.

I have verified this by numerous examinations of eyes with detached retinæ, which had to be removed for various causes.

In the light of the above description of this layer, it also seems probable that the hyaline excrescences (Glassdrüsen frequently noticed in pathological and even in otherwise healthy eyes are due to outgrowths of the gelatinous portion of the hexagonal layer.

(To be continued.)

CONGENITAL NIGHT-BLINDNESS AND PIGMENTARY DEGENERATION.

By Dr. COLMAN W. CUTLER.

(From the Clinic of Prof. Fuchs.)

(*With 2 lithographic plates, I. and II. of Vol. XXX., German Edition.*)

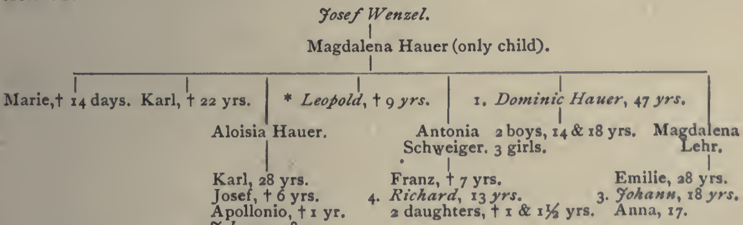
THE following cases of congenital night-blindness have been observed in the clinic of Prof. Fuchs in Vienna, and are reported at his suggestion.

The writer desires to express his gratitude for the opportunity offered him to use the material of the clinic, and for the assistance and advice so kindly given.

The first four cases have been known in the clinic for years, one member of the family having been examined by Prof. Arlt, but no records remain, except in one case, of previous examinations with which the present ones might be compared.

The mother of one of the patients, Case 2, is a nurse in the clinic; so it has been possible to get a complete history. and to examine three of the four repeatedly. Dominic Hauer, Case 1, the only one who does not live in Vienna, could only be examined once, but fortunately the record of the ophthalmoscopic examination, made by Prof. Fuchs three years ago, was at hand for comparison.

The following genealogical tree shows the transmission of the condition through five generations, affecting only males.



Those in italics are night-blind. * Was examined by Prof Arlt. † Dead.

Josef Wenzel, beyond whom the family traditions do not reach, was a mason by trade, living in Southern Austria. He was considered an unusually strong and healthy man, and died at about the age of eighty.

He suffered with night-blindness during his entire life, and it is believed that no change or progress took place in the condition. With a good light, vision was excellent. "He always went to bed at an uncommonly early hour, because his sight became poor at dusk."

Nothing is known of his ancestors, and consanguinity is denied.

To avoid repetition, it may be stated that no intermarriages have occurred in the family; no traces of syphilis are discoverable; there is no deafness; and no other diseases or anomalies of an hereditary nature have been found.

In the second generation there was only one child, Magdalena Hauer, whose husband is said to have been healthy.

In the third generation there were seven children; three males, two being night-blind.

Case 1.—Dominic Hauer, aged forty-six; mason; has been night-blind during entire life. The condition has remained stationary. The left eye has always been weaker than the right, and as long as the patient can remember has had its present position. General health has always been excellent. Patient is a well built man, of good intelligence. General examination negative. Color of hair, light brown; of iris, gray. Teeth show no signs of syphilis or rachitis. The left eye is deflected slightly upwards and outwards, the pupil being slightly covered by the upper lid; this is caused by the raising of the bulbus and not by ptosis. The eyes externally, otherwise normal. Pupils equal, of normal size, and react well. The ophthalmoscope shows the media to be clear. The right fundus somewhat tessellated, the retinal pigment being uniformly rather scanty, but not to a pathological degree. Vessels a little narrow, but not abnormally so. The upper temporal arteries are of good size; the lower relatively small. Professor Fuchs, who had the kindness to examine the patient, considers the difference in the size of the vessels so slight that it would not have been noticed had we not been especially on the watch for abnormalities. Temporal half of papilla slightly pale, shallow physiological excavation. Small conus outwards. No signs of pigment, and no white points or patches to be found in the fundus. Left eye the same, but with more uniformity of the vessels. The patient was examined three years ago by Professor Fuchs and it was seen that no change had taken place since that time, the same conditions of the vessels being noted at each examination.

Vision.—Right eye, $\frac{6}{12}$. Sn. 0.6 read at 12–38 *cm.* No astigmatism. Not improved by glasses. Left eye, $\frac{4}{60}$. Snellen not read. The light-sense was tested by Forster's photometer: An opening having a diagonal of 14 *mm* was required for the perception of light, and one or two more before the lines could be counted; $L = \frac{1}{49}$. [The normal eye requires an opening (h) of 2 *mm* diagonal; the opening required by the hemeralopic eye being H, light-sense, $L, = (\frac{h}{H})^2$.] The degree of night-blindness was the same after fifteen minutes and after one hour's adaptation. Color-sense, tested by the wools and by Stilling's test, normal.

The field of vision, examined by the perimeter, showed the outer limit, for white, of normal extent. The striking variation from the normal which the perimetric test with colors exhibited, will be discussed later to avoid repetition. Aloisia Hauer, the nurse in the clinic above mentioned, also belonging to the third generation, the mother of two of the patients examined, was found to have no hemeralopia. Normal fundus and field of vision. Of the fourth generation four were examined, three of them being night-blind.

CASE 2.—Johann Hauer, aged twenty-one, apprentice in book store. Among the usual symptoms of night-blindness he says that he has only seen the largest stars. There has been no change whatever in the condition, which was noticed, according to his mother, in infancy. He has always been near-sighted, but reads easily with a good light. The patient is of more than average intelligence. General health has been good. General examination shows nothing abnormal except slight enlargement of cervical glands. The teeth are bad, but not characteristic of rachitis or syphilis. Hair and iris brown. Pupils rather large, react well.

Vision.—Right eye, counts fingers at 4 metres. Sn. 0.5 at 8 *cm.* $-9\,D = \frac{6}{24}$. Left eye, $\frac{6}{60}$. Sn. 0.5 at 14 *cm.* $-9\,D = \frac{6}{24}$; with $-9\,D$ before both eyes he reads $\frac{6}{18}$. No astigmatism. Quick lateral nystagmus. Central color-sense normal. Light-sense : opening having a diagonal of 12 *mm*, $L = \frac{1}{36}$, after adaptation of 15 minutes and one hour. Ophthalmoscopic examination shows the fundus tessellated as in Case 1. Vessels perfectly normal. A large conus downward and outward from the papilla, which has an oval appearance. No pigment or white spots. No signs of choroidal disease.

CASE 3.—Johann Lehr, aged eighteen ; apprentice to hatmaker, gives the same history of night-blindness as the others. Fairly intelligent. General examination negative. Hair and iris dark brown. Pupils normal.

Vision.—Right eye, $\frac{6}{24}$. Skiascopy shows no refractive error. Left eye, $\frac{6}{18}$, $\frac{6}{12}$. Skiascopy shows slight hypermetropia. Both eyes, $\frac{6}{18}$. Sn. 0.5 at 10–25 *cm.* Ophthalmoscopic examination shows fundus to be very similar to Case 2. Vessels normal. A large conus downwards. No signs of pigment or white spots. Hemeralopia. A diagonal of 10 *mm.* $L = \frac{1}{25}$, after 15 minutes and after one hour adaptation. Central color-sense normal.

CASE 4.—Richard Schweiger, age thirteen years. General health good. Night-blindness as in the others, but seems to have been a little less marked. Fairly intelligent. Hair and iris dark brown. Teeth good. Pupils large, react well to light and accommodation.

Vision.—Right eye, $\frac{6}{36}$, with $-$ 2 D $= \frac{6}{18}$. Left eye, $\frac{6}{24}$, with $-$ 2 D $= \frac{6}{18}$. Sn. 0.5 at 5–20 *cm.* Central color-sense normal.

Hemeralopia, 12 *mm* ($L = \frac{1}{36}$) after adaptation of fifteen minutes and one hour. Ophthalmoscopically the fundus is tessellated, especially in the neighborhood of the papilla. A conus downwards and outwards nearly two thirds the diameter of the papilla, which is obliquely elliptical. Vessels normal. No deposit of pigment and no white spots seen.

Karl Hauer, brother of Johann, Case 2., is myopic ; with $-$ 9 D. Vision is $\frac{6}{18}$. No color-blindness. Field of vision normal for white and colors. No hemeralopia. $L = \frac{1}{1}$. In the fundus are several small spots of choroiditis in different parts, not in the macula.

This case is mentioned because it is the only one showing ophthalmoscopic signs of disease, yet the patient is entirely free from the subjective symptoms of the others, and also because it is stated that hemeralopia occurs in connection with myopia, and this patient being as myopic and as amblyopic as the others had no hemeralopia.

CASE 5.—Russian Jewess, age thirty-seven years. Sophie M——. Consulted Professor Fuchs with regard to symptoms of hyperæsthesia retinæ, and at the same time disclosed a condition which is unusually interesting. The family history is as follows: Her father lost an eye with glaucoma. The mother has had a cataract for fifteen years. A brother of the patient's father became gradually blind, and one of his daughters, aged fifteen, is blind, and, the patient thinks, was born blind.

The patient and two older sisters are night-blind. One is forty-two, the other forty years old. The night-blindness is in both congenital and has made no progress. Besides these there are three brothers and two sisters who have had no trouble with their eyes.

Madame M. has been married fourteen years and has three children living—one died at eight months with convulsions—who have had no symptoms of eye disease. No night-blindness.

One sister has had nine children, another three, all of whom are free from symptoms referable to the eyes. One brother has seven children free and another nine. The last married a second time, a cousin, and from this marriage came six of the nine children. The mother had external strabismus, as did her father to a still more marked degree, and three of the six children have it also. The grandparents were not related, and there is no knowledge of disease of the eye previous to the generation of the patient's father.

Several members of the family have died of phthisis. No symptoms pointing to syphilis can be made out. The patient has suffered with rheumatism for a number of years. At present she is hypochondriacal and somewhat hysterical, and complains chiefly, in addition to the hemeralopia, of being dazzled by bright lights and of headache and sense of fatigue after continued use of the eyes. These symptoms have varied with her general health. She is positive, however, that the night-blindness has been stationary. Ophthalmoscopic examination made by Professor Fuchs. Iris blue-gray. Pupils normal in size and reaction.

Right eye.—The papilla of normal color, sharply defined. Outward there is a rather well marked choroidal ring. The retinal arteries are distinctly narrow, the veins of normal calibre. The fundus otherwise normal, evenly red.

Left eye.—Outward from the papilla is a small atrophic conus which is not separated from the rest of the fundus by a sharp edge. The inner half of the papilla is bounded by a black, sharply defined choroidal ring. Vessels and fundus otherwise the same as the right eye.

Vision.—Right eye, $\frac{5}{12}$, + 1.5 D cyl. axis 30° nasal = $\frac{5}{6}$ + 4 D = Sn. 0.5 in 11 *cm.*

Left eye $\frac{5}{36}$, — 5.50 D spher. $\subset\supset$ — 0.5 D cyl. ax. 40° T = $\frac{5}{6}$, (?). Sn. 0.5 in 20 *cm.*

The photometer showed the degree of hemeralopia to be equal to 10 *mm* on the scale (L = $\frac{1}{25}$) after 20 min. adaptation.

It will be interesting to study the symptoms which are common to these five cases.

In all there is moderate night-blindness. The term light-sense is used here as Förster used it, namely, the acuity of vision in relation to the degree of light. In reality one gets a result with the photometer nearly equivalent to the least perception of light (Reizschwelle—irritation threshold), the exact determination of which is extremely difficult and clinically impracticable. The perception of minimum differences (Unterschiedsempfind lichkeit), does not enter into the question, if we may rely on the results of numerous examinations by Bjerrum,[1] who found that in all forms of night-blindness the perception of the slightest difference of light was normal. In three of the cases described above, the perception of difference was normal as compared with my own eyes. The other cases were not examined. The method used was a very simple one, based on the principle of Rumford's photometer. It was kindly suggested by Dr. Moritz Sachs, of Vienna. Two shadows of any broad object thrown upon a plane white surface by two candles of equal intensity, and at the same distance from the surface, are of equal density. If one candle is moved farther away or nearer, its shadows will vary in intensity. The movement which produces a difference between the shadows just perceptible to a normal eye, is the unit with which the pathological is to be compared.

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[1] *Graefe's Archiv*, vol. xxx., 2, p. 234.

The photometer, with a normal eye, required a diagonal of about 5 *mm* (L = $\frac{1}{6.25}$), without adaptation; after fifteen minutes' adaptation, L = $\frac{1}{1}$.[1]

As Schirmer says the degree of hemeralopia varies with the intensity of the preceding light for the normal unadapted eye, and also for the hemeralopic. The conditions were similar in these cases as regards the light, the examinations being made at the same time of day with the rooms moderately lighted, so it is fair to compare the results before and after adaptation, with the normal.

Schirmer[2] states that in hemeralopia due to various forms of choroiditis and retinitis pigmentosa and to glaucoma, after a sufficiently long time for adaptation, varying from one half hour to twelve hours, the sensitiveness to light reaches the normal. In the cases which I have examined, the sensitiveness to light improved as in the normal eye, the adaptation equalling about 3 *mm* of the Förster scale, in the first fifteen minutes, or less, and after that remained constant, not approaching nearer the normal. It was not possible to confine these patients for more than an hour to the dark room, but the results seem to show that the trouble depends on a diminished sensitiveness of the retina and not on the adaptation, which was apparently normal.

The central color-sense was normal in all of the five cases examined. The field of vision and the peripheral color-sense may be considered together.

CASE 1.—Whereas, with full daylight, the fields for white and red being normal and blue within the red, by diminished light, the field for white was not narrowed more than was the case in the check test; the colors, however, were in their normal relation to each other, blue being outside the red.

CASE 2.—With diminished light the outer limit for white was narrower than the normal, and the limits for colors still in their abnormal or inverted order. The field for yellow corresponded very closely to that for blue by daylight; with still less light, blue was lost first, red being still recognized.

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[1] Treitel, *Graefe's Archiv*, xxxi., 1, p. 153, gives for the normal unadapted eye L = $\frac{1}{49}$ or $\frac{1}{36}$. It is possible that the preceding light was brighter or that there was some difference in the apparatus.

[2] *Deutsche med. Wochenschrift*, 1891, No. 3.

CASE 3.—With good light, normal periphery for white, the colors being much narrower and inverted. With diminished light, white being disproportionately narrow, as compared with the check test, red had become slightly narrowed concentrically, so that the limits for red and blue lay nearly together. The answers in this case and in Case 4 were sometimes contradictory with the lower degrees of illumination, and in repeated examinations the results were not always exactly the same, but the essential points which I have mentioned were constant. With good light, the field for blue and yellow corresponded almost exactly; with the least light, yellow was generally seen as white.

CASE 5.—The field for white by a good light was normal, except for a very slight narrowing at the outer borders in both fields. Red was somewhat narrow. Blue and yellow within the red, with green nearest the centre. With somewhat diminished light, the field for white not being narrowed for the normal eye, in the case of the patient, the outer limits were slightly narrowed, and the limits for colors much more; in the right eye, red and blue falling together at 10° upward, outward, and inward, and at 7° downward. And for the left eye with the same light, the periphery for white was a little wider than the right, and the colors were narrowed concentrically almost to the fixation point.

With light still more diminished to a point just short of that with which the patient abruptly ceased to see the fixation point of the perimeter, the patient's peripheral perception of a 20 *mm* white object was $\begin{array}{ccc} 35 & 30 & 35 \\ 40 & \mathrm{R} & 42 \\ 50 & 45 & 45 \end{array}$, while the observer's with the same light was $\begin{array}{ccc} 50 & 50 & 55 \\ 50 & \mathrm{R} & 65 \\ 55 & 60 & 60 \end{array}$.

With this light the patient could not recognize colors at the centre; with slightly brighter light, red and green were recognized first, blue and yellow simultaneously with a degree of light still a little more intense.

These cases have in common, by daylight, normal field for white; normal or slightly narrowed for red. For blue and yellow—the colors falling almost together—margins within the red, with green in its normal position or perhaps a little narrowed. By diminished light, the periphery contracted in proportion to the light more rapidly, in all but Case 1 than in the normal eye examined under similar conditions. The fields for colors contracted more with diminished light than that for white.[1]

In Cases 1 and 3, with diminished light the colors regained their normal order. In Case 5 they were no longer inverted, but the fields for red, blue, and yellow coincided.

In Case 2 the order was still inverted.

I have found this inversion of the colors in several cases of idiopathic hemeralopia, with normal field for white, the colors returning to their normal order after recovery in two cases which were kept in the dark for several days.

In one case of retinitis pigmentosa in a young man whose parents were related but with no history of heredity, the margin for white was at 20°, and for red at 10°, with blue at 5°. In this case the pigmentation was very marked and characteristic, extending almost to the centre. The vessels small, and there was partial atrophy of the opticus.

Séggel[2] found this color anomaly in the case of two brothers, in whose family night-blindness had occurred during three generations. The field for white was normal, for blue diminished, falling with the red between 30° and 40°.

In both cases there were irregularities of the pigment in the fundus, and the arteries were small. Leber[3] alludes to its occurrence in idiopathic night-blindness; Mauthner[4] found a blue-blindness in glaucoma, which he associates with choroiditis of the macula region as opposed to the red-green defect more characteristic of optic-nerve disease. In glaucoma also, hemeralopia is a familiar symptom. Macé and Nicati[5] have found blue-blindness in icterus, and consider night-blindness to be a form of blue-blindness. In detachment of the retina[1] the fields for colors may be most irregular, but, as a rule, blue is badly seen, and often confused with green, and *vice versa*.

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[1] Leber (*Graefe-Saemisch*, v., p. 647) mentions the relatively narrower field for colors than for white, and ascribes it to the weaker intensity of the colors. In this sense it would be characteristic of all forms of hemeralopia, and not, as Treitel (*Arch. f. Ophthalmologie*, xxv., 3, p. 35) is inclined to think, a symptom peculiar to retinitis pigmentosa.

[2] *Bericht über die Augenkrankenstation des Garnison-Lazareth's*, München, 1884, p. 108.

[3] *Graefe-Saemisch*, v., p. 998.

[4] *Die Lehre vom Glaukom*, p. 190.

[5] Nagel's *Jahresbericht*, 1881, p. 143.

In the cases of congenital night-blindness which I have examined, the central color-sense has been normal, the defect occurring only in the periphery, and green and blue were not confused.

It would be a rash conclusion to draw from these instances, that night-blindness and blue-blindness are intimately associated, but their frequent coincidence is certainly suggestive.

The following case which I have examined recently is interesting in this connection.

Edward Urbanetz, aged fifty-three, painter. Family history negative. No consanguinity. The patient has eight healthy children. Fourteen months ago his house was burned, and he was greatly frightened and exhausted; he was in intense heat, but was not burned. At first he noticed nothing the matter with his eyes, but in a few days observed that he saw badly in a dim light; this has increased steadily and is now extreme. He does not think that his vision by bright light has deteriorated much. Formerly colors were seen and differentiated with accuracy as his trade required; since the beginning of his trouble, he says that he has had difficulty in telling the darker hues of green and blue apart in a poor light.

Patient denies having had syphilis, and does not drink or smoke to excess.

Examination shows aortic and mitral lesions, the liver is enlarged and tender, he has had œdema of the feet and probably ascites, but at present there is no fluid in the abdominal cavity. Color is sallow, the conjunctiva being slightly yellowish.

Vision, $+ 1.25\ \mathrm{D} = \frac{5}{18}$. Field of vision, right eye:

	Up.	U.O.	Out.	O.D.	D.	D. in.	In.	In up.
White......	50	70	70	80	60	45	55	60
Red........	15	35	35	30	12	20	15	15
Blue........	8	10	10	10	10	12	10	8

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[1] Dimmer, "Beiträge zur Pathologie der Netzhautablösung," *Wiener Medizinischen Presse*, 1886.

Outwards, blue was named green nearly ten degrees before being recognized as blue. Yellow followed blue quite closely. Green was seen only five degrees or less from the fixation point. Color test with Holmgren's wools showed no defect.

The field for the left eye was approximately the same.

The hemeralopia, without adaptation, with fifteen minutes, and after forty-five minutes' adaptation, was greater than could be measured with the Förster photometer (diagonal 50 *mm* $L < \frac{1}{625}$). The perception of differences of light normal, as compared with my own. Ophthalmoscopic examination showed the fundus to be normal, except that the arteries were a little small and the papilla perhaps a trifle pale. No pigment changes.

There is no xerosis of the conjunctiva. Tension normal. It is safest to associate the night-blindness and the peripheral blue-blindness with the disease of the liver, but the case will repay further observation.

Wolffberg[1] states, as a result of numerous examinations, that in all true hemeralopia, in choroiditis, and in separation of the retina, never in glaucoma, he has found the blue within the red. If the choroiditis was circumscribed, the color anomaly corresponded to the diseased area.

Unfortunately, he does not mention retinitis pigmentosa. Wolffberg associates it with abnormal conditions of the pigment epithelium. On the other hand, Raehlemann[2] states that in the atrophy of the optic nerve which accompanies retinitis pigmentosa, but which is found often where there is no pigment, with a dirty yellowish, blurred disc and narrowed vessels, the light-sense being diminished, he has discovered no disturbance of color-sense. Treitel,[3] in ten cases of retinitis pigmentosa, found no such anomaly as the one under discussion. He says that congenital color-sense anomalies occur frequently in retinitis pigmentosa. Blue was confused with green. In one case green was seen farthest in the periphery, with blue and red in normal relations to each other, while in another, with blue and red normal, green was narrowed considerably.

Treitel[4] states, finally, that in diffuse inflammation of the retina and choroid, as well as in intraocular neuritic processes, the outer limits of the fields for colors are normal, as a rule, and retain their normal positions in relation to each other, and quotes Leber and Schön and Stilling as having the same opinion.

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[1] *Graefe's Arch. f. Ophthalmologie*, xxxi., 1, p. 78.

[2] *Graefe's Arch. f. Ophthalmologie*, xxi., 2, p. 37.

[3] *Graefe's Arch. f. Ophthalmologie*, xxv., 3.

[4] *Loc. cit.*, p. 20.

Ophthalmoscopically, our cases have no pigment deposits in the retina. The pigment epithelium was normal. No white points or signs of choroidal disease.

In three cases, 2, 3, and 4, there was a large conus downwards, or downwards and outwards,—the congenital conus.

In two cases the arteries were narrow; in Case I. very slightly, and only in one part of the retina and in one eye. In Case 5 all of the retinal arteries were uniformly narrow.

In four cases, belonging to one family, there was decided amblyopia.

The cases of congenital night-blindness hitherto reported are few in number and not given in detail.

A brief consideration of the literature of the subject will be interesting.

In several of the cases the literature is not easily accessible, so I have taken the liberty of quoting at some length such cases as seem to present points of especial interest. Let me take this opportunity to thank Dr. Sédan of Toulon, Dr. Moore of Dublin, and Dr. Waller Zeper of Haarlem, to whose kindness the following notes are partly due.

Little need be said of the cases from the pre-ophthalmoscopic time—Richter, 1828; Cunier, 1838; Stievenart, 1847—which are interesting, but not available for statistical purposes.

Donders also, unfortunately, does not describe the ophthalmoscopical appearances of his case, reported in 1854, but Maes mentions it with his cases in which the eyes externally and internally appeared normal, distinguishing them from the other class which he describes, and which is clearly retinitis pigmentosa.

Donders.[1] Male, sixteen years. Hemeralopia is extreme. "In moderate darkness, where others still see the passers-by, he does not see them. By diminished light he was unable to see a white shirt front while I could still count fingers. . . . The torpor retinæ was strongest in the neighborhood of the visual axis, and perhaps existed only there. He remarked that often he had only seen a tree in the evening as he passed it and not directly in front."[1] This phenomenon was not present in the cases reported above. Father and two brothers were similarly affected. Three younger children are free. The condition was stationary and congenital.

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[1] *Nederland Lancet*, May and June, 1854.

Maes[2] examined a brother of Donders's patient, nineteen years old. Vision by good light normal. Both eyes externally normal. Ophthalmoscopically the media are clear and fundus normal. No abnormal narrowing of field by diminished light.

Maes also examined a schoolmaster with myopia. Fundus, except for conus, normal. Slight nystagmus rotatorius. Field not narrowed in diminished light. The patient was one of a family of eight, of whom two brothers are night-blind. It also occurs congenitally in the family of his aunt, his mother's sister.

Förster.[3] Male, fourteen years. Ophthalmoscopic examination, negative. Night-blindness, moderate in degree, congenital, not progressive. Field of vision by daylight and diminished light is normal. A brother is also affected in the same way. Consanguinity and heredity not mentioned.

Swanzy[4] gives the history of a family of ten, five brothers and five sisters, of whom three sisters and two brothers are night-blind. The condition was congenital and stationary. No history of consanguinity of the parents nor of heredity. Two brothers and one sister were examined, the conditions being similar in all, only differing in the degree of hemeralopia. One patient was slightly hypermetropic, another myopic, and the third emmetropic. No color-blindness. The ophthalmoscope showed nothing abnormal.

As in Donders's case, the night-blindness was strongest in the neighborhood of the visual axis.

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[1] The superiority of peripheral vision over central might suggest a central scotoma, except that patients with central amblyopia see better by diminished light, while here the reverse was true.

[2] II. *Jahresbericht der Utrechter Augenklinik.*

[3] *Ueber Hemeralopie und die Anwendung eines Photometers*, Breslau, 1857. The three doubtful cases mentioned by Förster are not congenital night-blindness, and if the definition which I shall give be allowed, they cannot be transitions-forms as Leber styles them, but must be otherwise classified.

[4] *Irish Hospital Gazette*, March 15, 1872.

A characteristic which occurred also in the cases examined by the writer is thus stated by Mr. Swanzy: "There is a point in the decreasing daylight up to which the patient can see well even to read the smallest type, but beyond which his eyes become suddenly almost useless to him."

Two of the sisters have, at different times, suffered with hyperæsthesia of the retina. One of them, who also had night-blindness, avoiding bright lights, and the others, without night-blindness "was laid up for a lengthened time with a severe attack of hyperæsthesia of the retina of that form, it would seem, which is unattended with defects of the field of vision but accompanied with subjective appearances of light and colors."

Fitzgerald.[1] Rev. Mr. ——, age thirty-five. Night-blind as long as he can remember. The condition has always remained the same. There were fourteen brothers and sisters, two still-born, most of the others died young, "water on the brain" being the cause assigned for the majority. Three sisters are living, of whom one is night-blind, as well as a distant cousin on the mother's side. "The patient always enjoyed good health except that he suffers from psoriasis palmaris." Myopia $\frac{1}{8}$. Field of vision and ophthalmoscopical appearance normal. Color-sense normal, except that in the right eye there was a crescentic-shaped portion at the inner side of the visual field, extending from the margin to within a short distance of the centre in which green appears brown and vermilion the same though somewhat darker. No consanguinity.

(The possibility of an inherited syphilitic taint suggests itself here, and it would have added to the value of the case if more than one member of the family had been examined.)

H. Pagenstecher[2] examined two members of a family with congenital night-blindness, and gives a genealogical tree showing the transmission through five generations.

Those affected were always males; eight out of thirteen. Consanguinity not mentioned. Night-blindness extreme. Vision myopic in one case, in the other emmetropic. Field of vision by diminished light disproportionately narrowed. Color-sense normal. Fundus appears normal. General health good.

Dr. Sédan[3] reports the case of an officer with marked night-blindness without ophthalmoscopic changes and without narrowing of the field of vision or anomaly of color-sense, and especially with no trace of retinitis pigmentosa. The condition was congenital, stationary, and there was no history of consanguinity.

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[1] *Irish Hospital Gazette*, Jan. 15, 1873.

[2] *Centralblatt für Praktische Augenheilkunde*, 1878.

[3] *Receuil d'ophtal.*, 1885, p. 675.

Genealogical tables are given of two families. In the first are seventeen members, seven females and ten males. Of these twelve were night-blind, namely, five females and seven males. In the second family, among eighteen members, five females and four males were night-blind.

Dr. Sédan has very kindly allowed me to state the history of these families since the publication of his article in 1885. This brings the transmission to the fourth generation in the first family with three boys, two of whom are night-blind. And in the second family in the fourth generation, three children not night-blind are added to the three given by the author, who were night-blind.

Leber[1] gives a detached case. Female, twenty years. Congenital night-blindness. Not mentioned whether stationary. No other members of family affected. Parents related. Field of vision by daylight free. Color-sense not mentioned. Vision, My. $\frac{1}{5}$. R. $\frac{20}{70}$, L. $\frac{20}{100}$. Ophthalmoscopic examination shows a posterior staphyloma, slight decolorization in the macula, with a few irregular dark spots. Optic nerve and vessels normal

Nettleship.[2] Male. Age sixteen. Night-blindness, probably congenital, not progressive. Vision $\frac{6}{18}$, not improved by glasses. Fields slightly narrow by lamplight. Ophthalmoscopical appearances perfectly normal. No family history of similar complaints. No consanguinity of parents.

Records of ten families have been given and of two isolated cases, making in all fifty-four cases, thirty-six males and eighteen females. Consanguinity is only mentioned once, Leber's case. In two of the families only males were affected. The transmission is through both male and female ancestors. In the family Wenzel which I have reported, the transmission was three times carried over two generations, the females being exempt.

In two families the condition has been transmitted through five generations with little diminution of its intensity. This suggests the interesting possibility of tracing the family Nougaret, reported by Cunier, the members of which are probably still to be found in the neighborhood of Montpellier, thereby establishing the identity of the condition by means of the ophthalmoscope.

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[1] *Graefe-Saemisch*, v., p. 650.

[2] "Some Forms of Congenital Amblyopia," Group 7, Case xxvi. *London Ophthalmic Hospital Reports*, vol. xi., part 4.

In addition to these typical cases, let me note three of a less definite nature.

Stanford Morton[1] gives the history of a family with hereditary night-blindness, transmitted through six generations. Only the males were affected. The two members examined had very narrow fields of vision. Fundus normal. One member of the family whom Mr. Morton did not examine had "progressive myopia, irregularly scattered pigment at the periphery, and night-blindness." This, with the narrow fields of vision, casts a doubt upon the nature of the condition, although the pigment is said to be not at all typical of pigmentary retinitis.

Heinrich Kornitzer, forty-two years, came to Professor Fuchs in 1881 with congenital stationary night-blindness.

Vision.—Both eyes, $\frac{6}{24}$. With weak concave glasses, $\frac{6}{18}$. Sight has not become worse. Eyes externally normal. Slight oscillating nystagmus, especially at the ophthalmoscopic examination. Fundus normal. There is concentric narrowing of field for white. Central color-sense normal. Photometric examination, $L = \frac{1}{35}$.

There are thirteen brothers and sisters. Four who are dead had good sight. Of the nine living three are night-blind—the patient, a brother two years older, and a sister fourteen years younger. The patient, as well as his brother and sister, have children whose sight is good. There is no consanguinity. Unfortunately it was impossible to trace this family; so that on account of the narrow fields of vision, in spite of the normal appearance of the fundus, it seems best not to assign them to any class because of the lack of evidence.

Night-blindness is a symptom occurring early in retinitis pigmentosa, possibly preceding the visible changes, but not constant, and not in itself sufficient to establish a diagnosis of that disease; occurring also *in choroiditis*, in glaucoma, in detachment of the retina, in various diseases of the liver, in pregnant women, after hemorrhage, as an acute disease, and, as is here assumed to be the case, as the result of a congenital malformation. It is hardly necessary to say that all of these causes, although acting on the retina in quite different ways, may produce the same effect.

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[1] *Transactions of the Ophthalmological Society*, 1893.

It is the conviction of the writer, though not at present susceptible of absolute proof, that this condition should be classed with the congenital anomalies or malformations rather than with the diseases, properly so called, resulting from inflammatory or degenerative changes.

The absolute unchangeableness, the association in several cases with the congenital conus and with marked amblyopia, and the fact that in none of the families reported is there a single instance of a variation of the type, which would certainly be the case if the condition were of inflammatory origin, are reasons for this view.

One meets the doubt frequently expressed as to the actual existence of this condition independently of retinitis pigmentosa.

Before the day of the ophthalmoscope night-blindness and torpor retinæ were all-embracing names. Since then, with the swing of the pendulum from the empirical and symptomatic to the exact and pathological extreme, most cases which have pigment in the retina, and many which have none, have been called retinitis pigmentosa. It is true that an exact classification either from a symptomatic or a pathological standpoint is at present impossible; but the vague, though often convenient, term atypical retinitis pigmentosa can and should be narrowed as advances in our knowledge give us clearer insight.

Retino-choroiditis, generally of syphilitic origin, has been more or less clearly differentiated. The retinitis punctata albescens of Mooren has a distinct identity.

Nettleship[1] describes a group of cases which clinically is very like pure congenital hemeralopia, except that the field of vision is somewhat contracted by daylight, and in all of the cases there was more or less marked retino-choroidal change.

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[1] *Ophthalmic Hospital Reports*, vol. xi., part 4.

A small but interesting group is also described by Nettleship[1] as probably the result of a retrobulbar neuritis forming a large and ill-defined central scotoma, having day-blindness, total color-blindness, and, in some of the cases, the ophthalmoscopic signs of retinitis pigmentosa.

More or less persistent night-blindness may follow acute infectious diseases, probably as a result of retinitis or retino-choroiditis, with very slight ophthalmoscopic change, as in the cases of Dor[2] and Hersing.[3]

One meets the statement that retinitis pigmentosa without pigment is not a rare occurrence, but I have been able to find only a few cases in the literature, which, for the sake of comparison, are given in tabulated form. In addition to these it is stated that Galezowski[4] has reported such cases, and Schmidt[5] states that one case of retinitis pigmentosa without pigment was seen in the Bonn clinic from 1883 to 1890; the case, however, is not given in detail.

It is probable that the earliest change visible with the ophthalmoscope in typical retinitis pigmentosa is the irregularity of the pigment epithelium. What precedes this we do not know. The nutrition of the deeper layers of the retina is perhaps in some way affected either through the choroidal or retinal blood supply.

The post-mortem examinations have been made upon eyes which are in an advanced stage of the disease, when it has lost what distinctive character it may have had and is indistinguishable from other forms of chronic retino-choroiditis.

Until pathological research or experiments on animals throw some new light on the subject we must view it from a purely clinical standpoint.

A conclusion that seems inevitable after reviewing the cases of retinitis pigmentosa and especially of congenital amblyopia is that the pigment changes are part of a general degeneration involving the retina, choroid, optic nerve, to

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[1] *Ophthalmic Hospital Reports*, vol. xi., part 4.

[2] Dor, *Arch. d'ophtalmol.*, 1883.

[3] Hersing, *Graefe's Archiv*, xviii., 2, p. 69.

[4] Galezowski, *Recherches ophtalmoscopiques*, etc., p. 22, Paris, 1863.

[5] Ernst Schmidt, "Über Retinitis pigmentosa." *Inaugural Dissertation*, Bonn, 1880.

	Pigment.	Vessels.	Optic nerve.	Central vision.	Peripheral vision.	Color-sense.	Hemeralopia.	Course.	Heredity.	Consanguinity.	
Darier, 1887. *Arch. d'opht.*, t. vii., p. 173. (Case 4.) Male, 18 yrs.	No pigment deposits in the retina.	Very small.	Dull red.	R. $\frac{1}{3}$. L. $\frac{1}{2}$.	Narrowed.	?	?	Probably progressive.	Grand-father blind at 30.	0	Case 4 is unquestionably retinitis pigmentosa without pigment. The diagnosis of the case alone would be probable, and it is made certain by the typical retinitis pigmentosa in the brother.
Darier. (Case 5.) Brother of Case 4, age, 31.	Choroidal atrophy around the papilla; a little pigment of typical shape in the periphery.	Small.	Dull red.	R. $\frac{1}{40}$. L. $\frac{1}{30}$.	$\frac{1}{3}$ narrower than normal.	Poor.	0				
Darier, *loc. cit.* Male. (Case 6.)	No typical pigment; 2 or 3 small black points along the vessels.	Very small.	Dull reddish gray.	R. $\frac{1}{3}$. L. $\frac{1}{10}$.	Very narrow.	Good.	Began at 5 yrs.	Gradually progressive.	0	0	The patient and two brothers, who died in infancy have six fingers and toes. A brother of his mother has a coloboma of the iris, and sees badly.
Hocquard & Poncet, *De la rétinite pigmentaire*, Paris, 1875. *Ann. d'Ocul.*, t. lxxiv, p. 234. Male, 21 yrs.	In one eye one pigment spot, in the other, 2 or 3.	Small.	Gray.	Poor.	Narrow.	Good.	Congenital.	?	Three ancestors.	?	Poncet found at the autopsy a good deal of pigment in the deeper layers of the retina, which had not been visible ophthalmoscopically, "because the retina was œdematous" (the patient had died of scarlet fever). There was also advanced endarteritis of the retinal arteries.
Treitel, *Graefe's Archiv*, xxv., 3, p. 20. Male, 18.	No pigment. Slight chorio-retinitis in macula. Small circular conus around papilla.	Very small.	Atrophic.	High myop.	White normal; colors seen only in a narrow circle around fixation point.	Has always seen blue badly.	Congen.	Not progressive.	0	0	Possibly a neuro-retinitis of *intra-uterine origin*.
Huidiez, *Annal d'Oculist.*, t. lxxviii., p. 211. Child, 8 years.	Infiltration at the border of papilla. No pigment.	?	?	?	Considerably narrowed.	0	Probably congenital.		A cousin is also night-blind.	Consanguinity exists in both cases.	It is a pity that the ophthalmoscopic examination is so indefinite. The term "infiltration" conveys no definite idea.
Alvarado, *Centralblatt f. prak. Augenheilkunde*, October, 1882, p. 312. Male, 31 yrs.	No pigment; retina orange red.	Small.	White.	Myop.	Narrow.	?		Progressive.	?	?	Post. polar cataract. There seems to have been no night-blindness, and the diagnosis cannot be considered as established.
Guaita, *Nagel's Jahresbericht*, 1875, p. 386.	No pigment; pigment epithelium thin at periphery.	Thin.	Pale.	?	Field narrow outwards.	?	Since 4th year.	?	2 Sisters night-blind.	?	Stated in Nagel, *loc. cit.*, that this case is congenital hemeralopia. It seems rather to belong to the class of retinitis pigmentosa, but the diagnosis could only be made positively after examination of the other members of the family.

speak only of the eye. And it seems probable that the disease may show itself in any one of the three places and retain the character of disease of that portion almost to the exclusion of the others, or they may be combined in varying degrees. If the name retinitis pigmentosa be used in this sense it could be made to include forms of disease with very diverse clinical appearances. If with Wagenmann[1] we assume that retinitis pigmentosa comes from the choroidal side through disease of the ciliary vessels or of the capillaries of the choroid, then the atrophy of the nerve and retinal vessels would occur as a secondary change, as after other forms of choroiditis,—choroiditic atrophy of Gowers,[2]—although the choroiditis itself may escape detection and may even be with difficulty discoverable *post mortem*.[3] This, then, would explain many of the cases of so called atypical retinitis pigmentosa where the characteristic subjective symptoms are present with atypical ophthalmoscopic appearances. It also shows how gradual and almost imperceptible the difference may be between retinitis pigmentosa and choroido-retinitis, making all transition forms possible.

If narrowness of the retinal vessels with hemeralopia but without visible ophthalmoscopical changes in disc or epithelium is also to be considered as incipient retinitis pigmentosa, then such a case as No. 5 would have to be classed with the group of retinitis pigmentosa without pigment, but this is not proven, and all we can say for the present is that the normal field for white, the stationary and congenital character and the otherwise normal appearance of the fundus, point more strongly to congenital hemeralopia than the narrow arteries do to retinitis pigmentosa.

All attempts at an exact classification must seem from certain points of view artificial.

It is not to be denied that underlying some of these classes the same inherent tendencies probably exist, predisposing to malformations and degenerative changes or low-

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[1] Wagenmann, *Graefe's Archiv*, xxxvi., 4., p. 1, and xxxvii, 1., p. 230.
[2] Nettleship, *Diseases of the Eye*, p. 191.
[3] Leber, *Graefe-Saemische*, v., p. 625.

Norm

Gr. f. W

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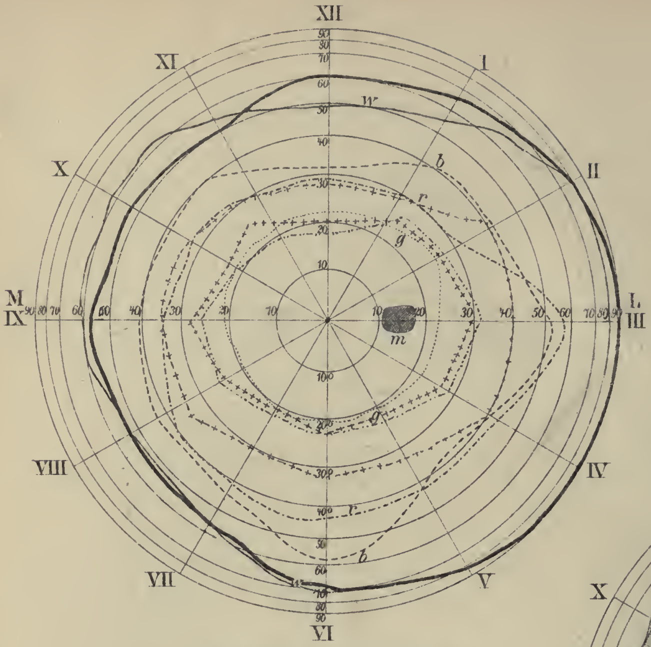

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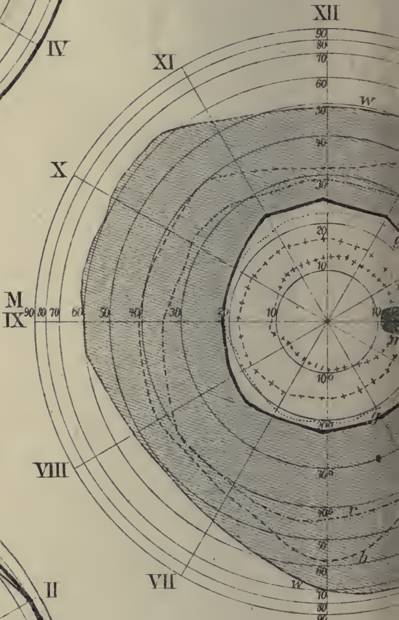

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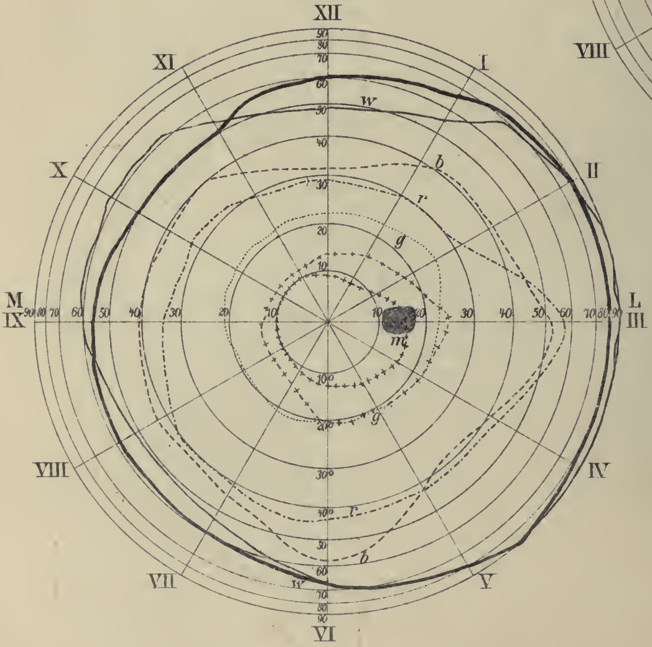

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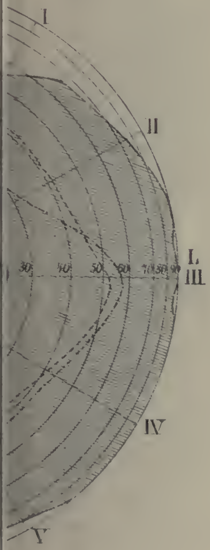

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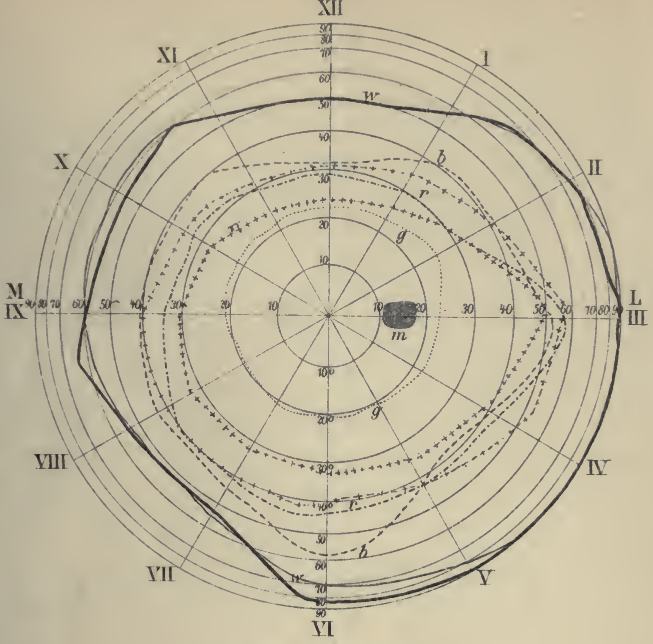

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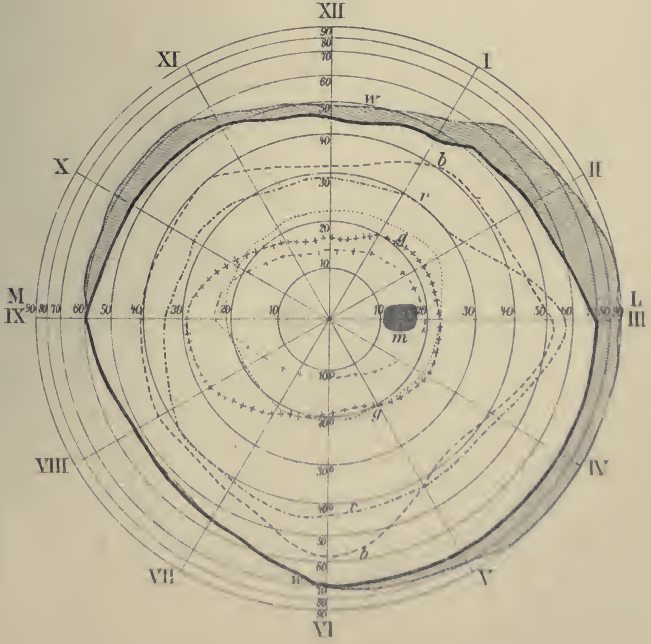

Fall Nr. I (bei schwacher Beleuchtung).

VERLAG VON J. F. BERGMANN, WIESBADEN.

grade inflammations. Of the determining causes we know nothing, and our conclusions can only be drawn from clinical observation and must be subject to future revision.

Finally, I have attempted to show that the night-blindness which is congenital, stationary, generally hereditary—and that to a greater degree than retinitis pigmentosa,—and not accompanied by signs of such disease as would in itself cause night-blindness,[1] is a condition depending upon a malformation of the deeper layers of the retina, probably of the pigment epithelium, the nature of which we do not know. Not being of an inflammatory or degenerative nature, there can be no question of transition or mixed forms between this condition and retinitis pigmentosa, such as are mentioned by Leber, Nuel, and others.

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[1] It is possible to conceive of congenital night-blindness being present in an eye in which retinitis pigmentosa afterwards develops, but this is a purely theoretical consideration.

THREE UNUSUAL CASES OF RETINO-CHOROIDAL DEGENERATION.

REPORTED BY COLMAN W. CUTLER, M.D.

(From the Clinic of Prof. FUCHS.)

(*With drawings by* Dr. SALZMANN. *Pl. III. Vol. XXX. Germ. ed.*)

THE following cases were seen by Prof. Fuchs while the writer was studying the subject of night-blindness, and the opportunity to report them was most kindly given him.

The patients are three sisters belonging to a Bohemian family. The father is dead. It is believed that there was nothing wrong with his eyes. The mother was examined and found to have normal eyes. The parents were first cousins.

No other hereditary or congenital form of disease exists in the family. There is no nystagmus.

Seven children are living; three dead, two with diphtheria at an early age, making it impossible to say what was the condition of their eyes.

Of the seven living, four are males, twenty-three, twenty, sixteen, and twelve years of age, respectively. All have normal vision.

The three sisters are twenty-six, twenty-four, and twenty-one years of age. They have in common night-blindness, cataract, and the peculiar form of choroidal atrophy at the periphery bounded towards the disc by a lobulated margin of the normal fundus.

There are no signs of inflammation.

Case 1.—Gisella Geyer, age twenty-six. Vision: Right, —18 D $= \frac{5}{24}$. Sn. 0.5 at 6 *cm.* Left, —20 D $= \frac{5}{24}$. Sn. 0.5 at 6 *cm.* Field of vision for both eyes considerably narrowed. The patient has three children, boys, with normal vision. She is a reddish blonde, the other sisters having brown hair.

In both eyes the iris is grayish blue. There is a small, almost round opacity at the post. pole of both lenses, without branches. There is no strabismus. The patient has been subject to fainting. The ophthalmoscope shows in the right eye (see Fig. by R. A. on Plate III.) a large staphyloma surrounding the disc, with peculiar lobulated protrusions, surrounded by normal fundus, and farther to the periphery a uniform zone of atrophy.

The edge of the staphyloma and of the atrophic zone are equally sharp in outline and partially marked with pigment. The fundus between the atrophic zone and the staphyloma is quite normal. The atrophic zone appears almost uniformly white, with a very few choroidal vessels near its inner margin; farther toward the periphery it is covered with a dense network of large, intensely black pigment, so thick that several parts of the periphery are almost black.

The left eye is nearly the same except that the branches or lobules of the staphyloma connect downwards with the peripheral atrophic area (see drawing Gisella G., L. A., Plate III.) The upper part of the fundus shows near the edge of the atrophic area several small round white patches, while in the other cases the atrophic areas are always confluent.

Case 2.—Irma Prohaska, age twenty-four. Vision: Right, perception of light at 1 *m*, outwards and inwards, not at the centre. Left, light perception, 5 *m.* Projection outwards and inwards good; upwards and downwards, uncertain. At eight years the patient could still go to school. At eleven she began to complain of night-blindness and to squint. She now has a high degree of external strabismus. For the past three or four years no change has been noticed in the vision. In both eyes, most marked in the right, there are many punctate opacities in anterior and posterior cortical layers, more numerous at the poles, forming there dense opacities.

The whole fundus shows atrophic choroid except at one place, outwards from the papilla. This place is two or three times larger than the disc, red, and surrounded by black pigment. In the central parts of the atrophic zone there is a little pigmentation of the fundus; towards the periphery are some larger masses of pigment, and some smaller deposits which are mostly branched.

The disc shows a high degree of atrophy. Right eye about the same, but because of the opacity of the lens the fundus cannot be distinctly seen.

CASE 3.—Rosa Prohaska, age twenty-one. Vision: Right, counts fingers at 2 *m.* Sn. 2.25 with difficulty. Field of vision considerably narrowed concentrically. Left, —10 D = $\frac{5}{60}$. Sn. 1.5 at 10 *cm.* Field slightly narrowed concentrically. For the past five or six years the patient has seen badly evenings and cannot venture in strange places at dusk. She reads in the shadow, when the book is well lighted, better than in the sun. There is marked external strabismus of the right eye. In both eyes posterior polar cataract in form of a delicate star with seven rays and darker centre.

Right eye.—The papilla is surrounded by a staphyloma which is continuous at one point with the peripheral atrophic zone, then follows the normal fundus as in Case 1, and the atrophic choroid for eight or ten papilla diameters to the periphery.

The outer or atrophic zone shows towards the normal fundus most of the larger vessels of the choroid, towards the periphery it becomes more and more uniformly yellowish and shows the vortex veins with unusual distinctness. The pigment epithelium has disappeared and there are round and branched and in some places very large and intensely black spots in this area. The disc is slightly atrophic.

Left eye.—Color of disc less atrophic than right. Large staphyloma outwards, nowhere connecting with the peripheral atrophic zone (see Fig. Rosa P. on Plate III.), which is not quite so large as in the right eye, but otherwise the same.

The right iris is grayish blue, the left the same, with some brownish pigment spots.

A similar case has been reported by Jacobson,[1] under the name of atypical retinitis pigmentosa.

The patient was a man of twenty years. The fundus was comparatively normal for a distance of two or three papilla diameters from the disc, this being bounded by a lobulated, somewhat pigmented, sharply defined margin, outside of which the choroid is atrophic nearly to the limit of the visible part of the fundus, but less completely so at the extreme periphery, being there interspersed with patches of normal fundus. Optic nerve pale, vessels narrow. In the atrophic zone and extreme periphery numerous pigment deposits along the course of the vessels, such as are characteristic of retinitis pigmentosa. The condition had existed since childhood and was progressive. Night-blindness marked. Field of vision concentrically narrowed. Color-sense normal. The patient was mentally somewhat deficient. The father of Jacobson's patient had been blind for a year and a half and night-blind since childhood. Ophthalmoscopically there were signs of a typical retinitis pigmentosa with white areas in the periphery as in the son, but no continuous zone of atrophy.

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[1] Zehender, *Monatsblatt*, 1888, p. 202.

Mauthner[1] has described a case of almost complete absence of the choroid, which deserves mention in this connection.

Male, thirty-two years. Has always seen badly and has been night-blind since childhood, but the condition has grown worse in the last fifteen years, since an attack of typhoid fever. Vision, R. $\frac{5}{200}$, My. $\frac{1}{12}$. Field concentrically narrowed, temporal side absent, greatest dimension vertical 20°. L. V. $\frac{10}{40}$, My. $\frac{1}{16}$. Field failing downwards, greatest dimension 34 ° horizontal. The ophthalmoscope showed in both eyes the fundus glistening, greenish-white, optic disc round, margins indistinct, retinal vessels somewhat narrow, otherwise normal.

The fundus shows only a few normally shaped choroidal vessels such as appear in colobomata, and small masses of pigment. Only in the macula is seen a net of choroidal vessels, while in the left eye this place is marked by a sharply defined reddish-brown, rhomboidal area. Here the pigment covers the vessels which are first visible farther out.

Of this case Leber[2] says: "The case of Mauthner described as choroideremie may be pigmentary atrophy of the retina complicated with extreme atrophy of the choroid."

We have, then, five cases, or with Mauthner's, which, from a certain point of view, belongs to the same group, six, which may be considered together and which give a fairly complete picture of the form of disease.

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[1] *Berichte des Naturwissenschaftlich-Medizinischen Vereines*, Innsbruck, II. Jahrgang, 2 u. 3 Heft p. 191.

[2] Nagel's *Jahresbericht*, 1872, p. 316, and *Graefe-Saemisch*, v., p. 642,

The older of Jacobson's patients is blind with retinitis pigmentosa, but the choroidal atrophy is slightly marked; his son is not blind, but the choroidal atrophy is more advanced and the condition is stated as progressive. These must be taken as less markedly atypical cases, not less advanced retinitis pigmentosa. In other words, we cannot look on the degree of atrophy as representing different stages in the development of the disease.

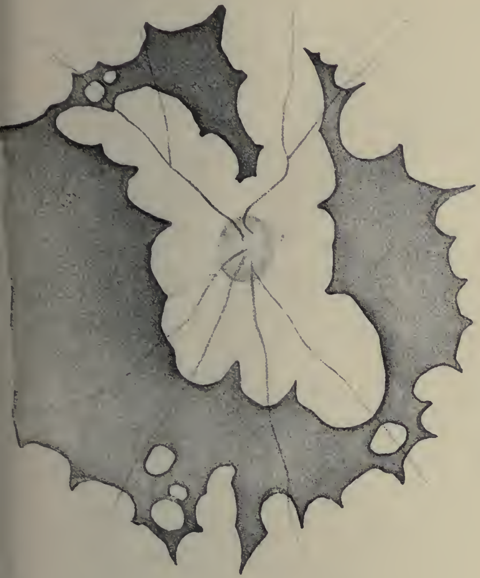
In logical succession follow Cases 1 and 3. In the former, in addition to the zone of total atrophy there are a few isolated patches of choroidal atrophy.

In Case 2 the choroidal atrophy is almost complete, the nerve is atrophic, and vision is almost abolished. If finally we take Mauthner's case of "choroideremie," we have a striking group which may be taken to represent different phases of the same condition. There is no evidence to show that they represent different stages of the atrophy. On the contrary, the impression given is that of a process which has run its course and reached a standstill.

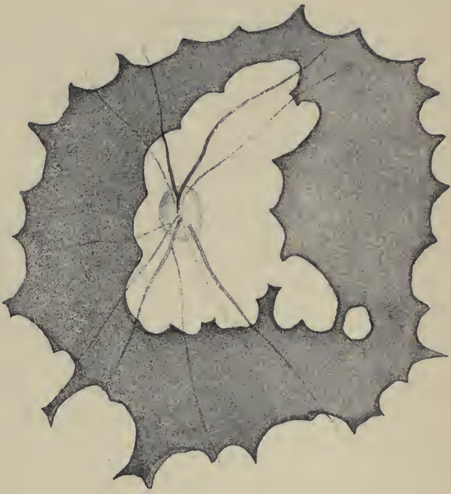
The sharply defined and pigmented edge and the absence of signs of inflammation would point to the stationary character, while the lobulated edge and the occurrence in two cases of isolated patches would indicate that the choroidal atrophy was formed by the growth and confluence of such smaller areas.

If we assume that the atrophic condition is stationary, what can be said of the retinitis pigmentosa and of its association with the choroidal atrophy? That the retinitis pigmentosa is progressive must be assumed from the nature of the disease. In thus dissociating the two conditions we weaken the idea of unity which seemed the most plausible explanation, namely, that both the choroidal atrophy and the retinitis pigmentosa are manifestations of the same disease though occurring independently.

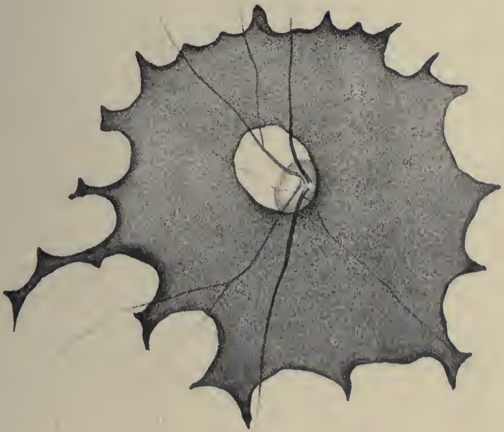
It is possible that in spite of the stationary appearance of the choroidal atrophy it may still be progressive either in a degenerative way without signs of inflammation or inter-



Gisela G. L. A.
Umgekehrtes Bild.



Gisela G. R. A.
Umgekehrtes Bild.



Rosa F.
Umgekehrtes Bild.

AD NAT. D. DR. M. SALZMANN.

ERSITÄTSDRUCKEREI VON H. STÜRTZ, WÜRZBURG. VERLAG VON J. F. BERGMANN, WIESBADEN.

mittently, and in favor of this view must be considered the fact that in all of the cases the essential features of the atrophy were the same. This assumption suggests the possibility of our cases eventually reaching the condition of complete atrophy which Mauthner's shows, but this is purely an hypothesis.

With regard to the vision, these cases present problems which are equally hard to solve.

We may eliminate the cataract as the least important element, the remaining ones being the retinitis pigmentosa, with the optic atrophy and the choroidal atrophy, in that order.

The fields of vision, unfortunately, could not be taken with sufficient exactness to throw much light on the subject, but it is obvious that light perception was present in a part of the field corresponding to the atrophy. The myopia is probably secondary to the amblyopia and perhaps to degenerative changes, and the staphylomata may also be progressive, advancing to meet the peripheral atrophy.

It is possible only to touch upon the details of this complicated picture and our ignorance of the true nature of the disease is as complete as ever. We can only consider it a form of that large class of pigmentary degenerations, affecting the choroid to an unusual and excessive degree.

In closing, let me express my warmest thanks to Dr. Salzmann for the drawings of the fundus which he has allowed me to use and for his kindness shown in many ways.

A STRAIGHT ARM, CORRECTED SCALE, AND OTHER IMPROVEMENTS FOR JAVAL'S OPHTHALMOMETER.

By CARL WEILAND, M.D.,

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(*With 3 drawings in the text.*)

IN the October number (1894) of the *Annals of Ophthalmology and Otology*, Dr. G. W. Grove publishes an article entitled "Graduation of the Arc on Javal's Ophthalmometer; and Graduation on a Straight Arm." In this he does not claim any advantages for a straight arm over the arc, but rather admits its inferiority (page 366). And indeed that is quite correct for his procedure, because he simply projects the old graduation from the arc to the straight arm, which must necessarily give inaccurate results, as the transfer of the mires from the arc to the straight bar changes the whole calculation upon which the arc was graduated. But I advocate the superiority of the straight arm over the arc. Dr. Grove refers to an article of mine in these ARCHIVES (Jan., 1893), in which I state in a note that the arc of Javal ought to be replaced by a straight line. I did not at that time express any further opinion about it and have not published anything about the subject up to this time. But I was just then finishing my calculations for a straight arm, which I have employed since the spring of 1893,[1] making at the same time such mechanical changes that both mires moved equally in opposite directions. The reason I had for introducing the straight arm was, first, because then the mechanism for the equal and opposite movement of the mires could be constructed more easily, and secondly and *mainly* because I wanted to increase the accuracy of the instrument, not for the purpose of prescribing cylinders but to make the corneal measurements as accurate as possible. However I did not simply project the graduation from the arc to the straight arm as Dr. Grove does, but I made new calculations altogether; because I objected to the use of an arc as being wrong in principle as will be shown now. Javal makes his calculations from the formula $\frac{1}{b} = \frac{1}{d} + \frac{2}{r}$ where the meaning of d, b, and r will be at once clear by reference to Fig. I. If further O is called the size of the *arc* $A\,B$, the image of which is the small *arc* $C\,D$ and is called J, we have $\frac{O}{J} = \frac{d+\frac{r}{2}}{\frac{r}{2}}$ Now Javal calls $d + \frac{r}{2}$ a constant, and uses it as such in his calculations. This of course is not quite correct as $\frac{r}{2}$ is variable; but the error does not amount to much and may be neglected from the present point of view.

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[1] At that time I had the pleasure of showing this modified instrument to Prof. H. C. Chapman and Prof. W. Thomson of the Jefferson Medical College of Philadelphia.

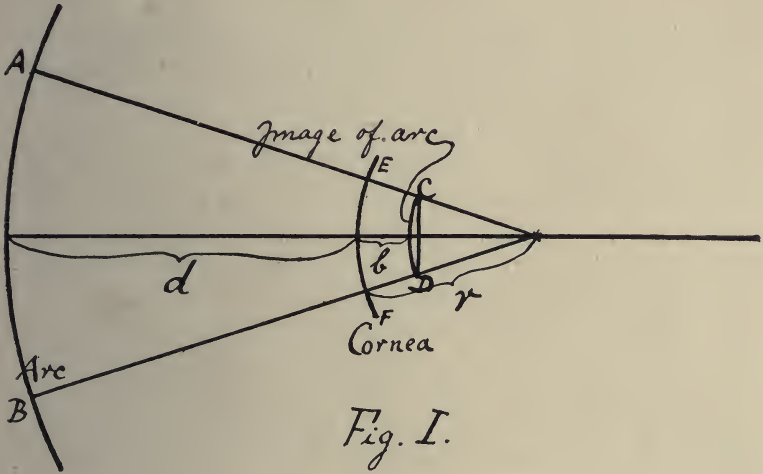

Fig. I.

However, the following inaccuracy is of much greater weight and cannot be accepted for accurate work. It arises from the use of an arc. It is true that a circular object, like an arc, is the only object that gives a circular image on a spherical mirror; but it is just this fact which is a source of serious inaccuracy in the instrument. For the Wollaston prism gives a doubling of a certain fixed quantity, in my own instrument equal to 2.92 *mm*, and this is not measured along an arc, but on a chord. The *chord C D* (Fig. I.) alone is fixed by the arrangement of the lenses and prism, but not at all the *arc C D*, which by calculation may be easily shown to vary from 2.94 *mm* for a corneal radius of 10 *mm* to 3.03 *mm*, for a radius of 6 *mm*. In the formula, therefore, which is used by Javal $\frac{O}{J} = \frac{d+\frac{r}{2}}{\frac{r}{2}}$, the quantity J is not fixed as he assumes, but is variable within 0.1 *mm*. Such a variability is not allowable; for the corneal image must be accurate within $\frac{1}{100}$ of a millimetre, as Helmholtz[1] says: "For the purpose of measuring the cornea one can only use images, which are considerably smaller than the corneal radius, not larger than one quarter of the size of the latter. One must endeavor to precisely determine these images to *the hundredth part of a millimetre*, if one wants to calculate accurately the corneal radius to the $\frac{1}{200}$ of its size." This applied to a radius of 8 *mm* would mean that the radius under these precautions would be accurate to $\frac{4}{100}$ of a *mm*. And that is not hair-splitting, because it corresponds to a quarter ($\frac{1}{4}$) of a dioptry, which we all like to get accurately. Thus it has been shown that Javal's instrument in its present construction cannot give us exactly the quarter of a dioptry.

It is for this reason that I have given up the arc and have substituted for it a straight arm. It is undoubtedly true that this straight bar has not a straight line for its image on the cornea. But that does not concern us here at all. All we have to consider is the place where the inner edges of the two mires have their images on the cornea—that is, how far these images fall—from the axis of the instrument. This is all that is necessary for our purpose, because if we then call O the straight distance between the inner edges of the two mires, which in our construction is perpendicular to the axis of the instrument, and if we call further J the distance between the two images, which is also measured at right angles to the axis, we have then conditions which allow of a rigid calculation, as *J* remains always the same. And, further, I did not only thus make *J* a fixed quantity, but I also employed formulæ which are more accurate than Javal's; for we must remember that the formula, before given, is not quite correct, if the object is very large with reference to the convex mirror and so near to it. What really happens can be perhaps best explained by reference to Fig. II. Here GH represents the cornea and AE the straight bar, while C is the centre of curvature of the cornea. If now A is a point on the straight arm, it is clear that only that ray from it can be seen by the observer which is reflected from the cornea at B into the telescope at E, in such a way that the angle of incidence ABH equals the angle of reflection HBE, called i in the diagram. All the other rays emanating from A and reflected from other parts of the cornea will not be seen by us, as they do not enter the instrument at E.

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[1] *Graefe's Archiv f. Ophth.*, i., 2, 1854.

Now, it can be shown that each point A has in reality two images, or rather two focal lines on a reflecting curved surface, which are found by the following two equations:

$$\text{(I.)}\ \frac{1}{b} = \frac{1}{d} + \frac{2\ \cos i}{r} \quad \text{and} \quad \text{(II.)}\ \frac{1}{b_1} = \frac{1}{d} + \frac{2}{r_1 \cos i}$$

Here (I.) gives the focus for rays from A near B vertically to plane of paper, while (II.) gives the focus for rays from A that fall near B in the plane of the paper. The letter r denotes the radius of curvature of surface B vertically to the plane of the paper and r_1 the radius of curvature at B in the plane of the paper, this plane being supposed to be one of the two planes containing the two principal radii of curvature of the surface at B. This shows, then, that for very astigmatic or ellipsoidal surfaces, the difference between the two principal radii of curvature ought to be taken into account; but it is also seen that even for a spherical surface, where r and r_1 are alike, we get two focal lines, which are, however, lying so near to each other, that practically they may be considered as coinciding in mirrors of corneal dimensions.

We take here the formula (I.), because then the construction and calculation are so much easier. It will be seen at once that now d and b have a different meaning from what they had in the usual formula for central rays, and it is evident also that on the straight bar $A\,B$ ($= d$) is not equal to $E\,B$. This, indeed, is the case in Javal's arc, which therefore, in so far, would deserve the preference, if the variability of $\mathcal{J}$ was not against it. One might, however, avoid the variable $\mathcal{J}$ by calling O the distance of the mire from the axis of the telescope and then proceeding in the same way as was indicated before for the straight arm. This would also change the scale of the arc, but even that would not be quite correct, as it can be demonstrated that then for different O and r the arc of the instrument ought to be of different length. The calculation to prove this cannot be given here for want of space, but it rests in a great measure on the fact that, in Javal's instrument, $E\,D$ (Fig. II.) is a constant, on account of the fixed position of the lenses, owing to which the image can only appear clear when $E\,D$ is of a certain size. But if the straight arm is employed, the objection to the change of arc falls away, though the calculation is then more tedious. I make now use of the correct formula (I.),

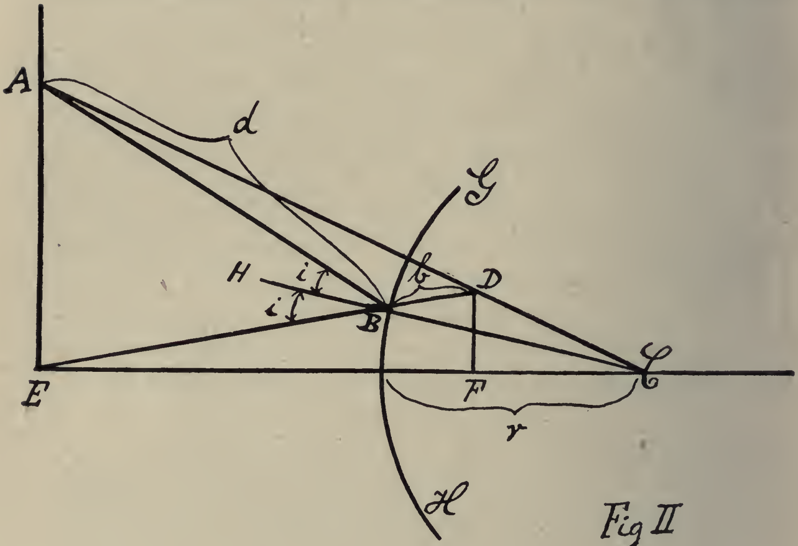

Fig II

$\frac{1}{b} = \frac{1}{d} + \frac{2 \cos i}{r}$ where d, b, and r have the meaning indicated in Fig. II, and of the formula $\frac{O}{\mathcal{J}} = \frac{d}{b}$, which follows from this if $\frac{O}{2} = A\ E$ and $\frac{\mathcal{J}}{2} = D\ F$ of Fig. II.

I shall now shortly indicate the most important steps in the calculation. First, we must remember that in every case we have two constants, the one being $E\ D$, which we will call δ, and the other being $D\ F$, or half the image $\mathcal{J}$, which are both determined by the construction of the lenses and the Wollaston prism. It will, of course, be understood that the diagram represents only one half of the straight arm and of the image $\mathcal{J}$, the remaining part being just the same on account of the equal but opposite movement of the mires of my own instrument. If one now proceeds to find under these circumstances the radius r, that belongs to a certain O, while $\mathcal{J}$ and δ are always the same, one comes to very long and awkward formulæ, which will not be given here. It is, however, comparatively easy to find the radius r by construction, as follows:

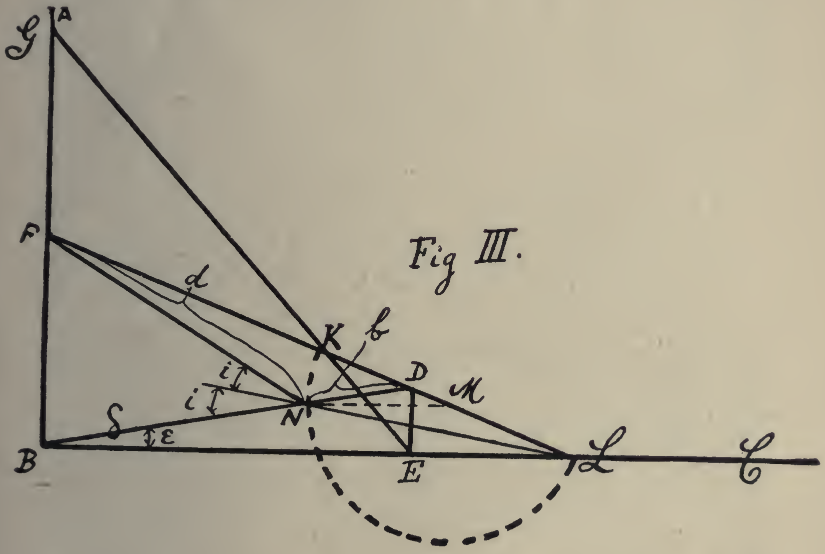

Fig III.

Let, in Fig. III, $A\ B$ be the straight arm, $B\ C$ be the axis of the instrument, on which also lies the unknown centre of the cornea. Now, first construct from $B\ D = \delta$ and $\frac{\mathcal{J}}{2} = D$

E, the right-angled triangle $B D E$. Let, further, $B F$ be the object $\frac{O}{2}$, the image of which shall be $\frac{J}{2}$, or $D E$. Now, make $F G = F B = \frac{O}{2}$ and draw $F D$, which may intersect $B C$ in L. Draw then $E G$, and call K the point of intersection with $F L$. Now bisect $K L$ and let M be the point thus found. Draw a circle from M with $K M$ as radius, and let this circle intersect line $B D$ or δ in N. Finally draw $F N$ and $N L$. *Then $N L$ is the radius of the cornea*, which latter will reflect ray $F N$ from the cornea into the telescope. The demonstration that this construction gives accurately $\frac{1}{N D} = \frac{1}{F N} + \frac{2 \cos i}{N L}$, or $\frac{1}{b} = \frac{1}{d} + \frac{2 \cos i}{r}$, and that $\frac{O}{J} = \frac{d}{b}$ would be too long, but can be easily supplied, if it is considered that $F L$ is harmonically divided. To find, now, r numerically, the following calculations must be made (Fig. III.):

(1.) $\sin \varepsilon = \frac{J}{2\delta}$ (2.) $\operatorname{tang} F L B = \frac{O - J}{2\delta \cos \epsilon}$

(3.) $F D = \frac{O - J}{2 \sin F L B}$ (4.) $K D = \frac{J F D}{O + J}$

(5.) $D L = \frac{J}{2 \sin F L B}$ (6.) $N M = \frac{K D + D L}{2}$

(7.) $D M = \frac{D L - K D}{2}$ (8.) $\sin D N M = \frac{D M \sin (F L B + \epsilon)}{N M}$

(9.) $\sphericalangle D M N = \sphericalangle F L B + \varepsilon - D N M.$

(10.) $\frac{r}{2} = N M \cos \frac{D M N}{2}.$

Thus for each O the corresponding r was calculated for my own instrument (a Javal of the old model), in which there was found $\delta = 342$ *mm*, and $\frac{J}{2} = 1.46$ *mm*.

The following table gives the result of this calculation:

Size of object *O*.	Difference in *O* for succeeding dioptries.	Dioptries of cornea.	Radius *r* of cornea.
221.66 *mm*	....	36 *D*	9.34 *mm*
238.32 *mm*	6.66 *mm*	37 *D*	9.09 *mm*
235.02 *mm*	6.70 *mm*	38 *D*	8.85 *mm*
241.76 *mm*	6.74 *mm*	39 *D*	8.62 *mm*
248.54 *mm*	6.78 *mm*	40 *D*	8.41 *mm*
255.37 *mm*	6.83 *mm*	41 *D*	8.20 *mm*
262.25 *mm*	6.88 *mm*	42 *D*	8.01 *mm*
269.18 *mm*	6.93 *mm*	43 *D*	7.82 *mm*
276.16 *mm*	6.98 *mm*	44 *D*	7.64 *mm*
283.20 *mm*	7.04 *mm*	45 *D*	7.47 *mm*
290.30 *mm*	7.10 *mm*	46 *D*	7.31 *mm*
297.46 *mm*	7.16 *mm*	47 *D*	7.16 *mm*
304.68 *mm*	7.22 *mm*	48 *D*	7.01 *mm*
311.96 *mm*	7.28 *mm*	49 *D*	6.86 *mm*
319.30 *mm*	7.34 *mm*	50 *D*	6.73 *mm*
326.71 *mm*	7.41 *mm*	51 *D*	6.62 *mm*

This means, then, that for a distance in a straight line of 276.16 *mm* between the inner edges of the two mires, the corresponding radius would be 7.64 *mm*, whilst the corneal image would measure 2.92 *mm*. This would show a refractive power of the cornea of 44 D. For 45 D the distance would have to be 283.20 *mm*, or 7.04 *mm* more than before. Now if both mires move equally in opposite direction, it is evident that each mire will move only through 3.5 *mm* to indicate one dioptry. If one, therefore, graduates the straight arm itself, one cannot read off as well, the spaces between two succeeding dioptries being only one half the size they were, when only one mire moved. To obviate this difficulty, and to make the reading as easy as before, I added a second graduated bar below the straight arm. This bar is fastened to one mire and slides in a little case below the second, where the dioptries are read off. In this way the new scale moves twice as quick as each mire, *i. e.*, with reference to the second mire, thus giving again 7 *mm* space from 44 to 45 D.

I further discarded the stepped mire altogether, because now these steps would be quite inaccurate, and besides it is always more accurate to produce contact in each meridian. I replaced the mires by two yellow squares of exactly the same size. The yellow color was chosen because the blue rays, that interfere most with the exact contact, are then quenched to a great extent; and the mires were made of the same size in order to enable us to measure the angle α. For this purpose the straight arm has been divided into degrees, and has also been provided with little sights that move along it. After finding one main meridian, the patient is told to look at one of the two sights, and this is moved until the two adjacent images of the two mires are of exactly the same size, because then the corneal apex lies on the axis of the instrument. Now the angle α can be read off directly and then the corneal astigmatism be determined.

In conclusion, I must say that the straight arm with the new calculation on the lower movable bar, the two movable yellow mires of the same size, and the division into degrees for the angle α, are, in my opinion, a decided improvement upon Javal's instrument as far as an accurate measure of the cornea is concerned. But of course I do not claim to thereby come much nearer to the cylinder to be prescribed; for this cylinder depends also upon the remaining astigmatism of the dioptric apparatus, on the axial ametropia, and the position of the glass in front of the eye, as has been explained before in these ARCHIVES, in the January number of 1893.

HOW JAVAL'S KERATOMETER MAY BE EASILY CHANGED INTO A GOOD CHROMATOMETER FOR THE EXAMINATION OF PATIENTS AS TO COLOR-BLINDNESS.

By CARL WEILAND, M.D.,

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FOR the examination of patients as to color-blindness, certainly the most convenient and practical tests are those that make use of colored worsteds, like Holmgren's or Dr. W. Thomson's test, and others. There is, however, one disadvantage connected with worsteds, namely, that they are all more or less apt to change in color, not only by handling but also by light, dust, and other circumstances. Many attempts have therefore been made to find a substitute for these wool skeins. The spectral colors, though theoretically the best, cannot be well employed in an office in spite of Hirschberg's double spectroscope and the excellent apparatus of Captain Abney. For these reasons another method has been employed which also gives unchangeable colors, though these are not the simple colors of the spectrum, but color mixtures like those of the worsteds. These colors are produced by allowing polarized light to pass through a quartz-plate and again through a Nicol's prism. *Rose* was the first to make use of this method in his *Color-Measurer*, which he described in *Virch. Arch.*, xxviii., p. 30 *et seq.* He causes the light to pass through a Nicol's prism first, and then by a diaphragm through a double refracting prism, from where it enters first a quartz-plate, cut at right angles to its optic axis, and finally a second Nicol. He thus produces two circles of complementary colors, that change continually when the upper quartz and Nicol are rotated, but always remain complementary to each other. The second instrument of this type was brought forward by *König* in his *Ophthalmo-Leukoscope* in 1884. It is like Rose's instrument, only differing in so far that the first Nicol is wanting, and that he uses quartz-plates of different thickness—5, 10, or 15 *mm* thick, according to the degree of saturation that the colors shall attain. The latest instrument of this kind is *Chibret's Chromatophotoptometer* of 1885. He uses, however, a quartz-plate that is cut *parallel* to its optic axis and obtains the change in colors by inclining the plate at different angles to the line of vision. These instruments have all their special advantages, but they are very expensive, and those of Rose and Chibret, in their present form, have that disadvantage that the color-fields are very small and must be looked at with about 4 D of accommodation. It occurred, however, to the writer to make use of Javal's keratometer, as this instrument contains already a very fine double refracting prism, and gives a considerable magnification. He got up a little contrivance of his own two years ago, which has worked so well since, that he thought himself justified in making it known to the profession, and in thus increasing the usefulness of Javal's instrument. His method differs from that of the authors mentioned above, though the principle is the same.

This color-attachment to Javal's keratometer consists of a straight metal tube, about 1½ inches in diameter, that reaches from the place where the patient's cornea usually is to about the beginning of the barrel of the telescope, and is so fastened to the head-rest that its axis coincides with the axis of the instrument. At the front part of this color tube there is a plane glass plate behind which a Nicol's prism is fastened in a cork. From this prism the polarized light passes by a round diaphragm through a quartz-plate, cut at right angles to its axis and about 5 *mm* thick.

This is essentially the whole outfit; for in looking with the Javal through this tube one will see two fine large color-fields partially overlapping each other. These color-fields are of complementary hues while the place of overlapping shows white; provided of course that white light as reflected from a white surface, like a piece of white paper, is employed in this experiment. If now the arc of Javal be rotated, while the color tube remains in the same position, the colors will change continually but always remain complementary, returning however to their original hues after the arc has been rotated through 90°.

I would recommend the instrument to be used in the following way: Place your patient's eye at the ocular of the instrument after you first have looked in yourself and given to the new color-tube such a position that blue and yellow appear, because thus most color-blind people will recognize two different colors. Now ask your patient whether the two colors are exactly alike or at least shades of the same color. If he answers *No*, turn the barrel of your Javal slowly through 90°, telling your patient to stop you as soon as the two colors are the same. If he has good color-sense he will always see two different colors, but if he is color-blind he will find that in a certain position of the arc the two colors appear alike or at least as much alike as if they were shades of the same color. These colors will usually be green and rose for a green-blind person, while the red-blind person generally selects a more bluish green and a rose with much more red in it.

This suffices to prove that your case is color-blind. But if you want to go further, if you wish that the two colors shall appear *exactly alike* even in brightness (what can never be obtained with worsteds), then take a second Nicol's prism and fasten it so in front of the eye-piece that it can be rotated and is as near as possible to it. If now your color-blind patient looks again, leaving your arc of Javal as before where the colors appeared almost the same, you will, on rotating your new Nicol, easily find a position where the two colors look exactly alike, in hue as well as in brightness. Now put down in your case-book the position of your color-tube, that of the arc of Javal and that of the second Nicol, and you will always be able to reproduce the same color equation, even after years, when the wools would long have faded. The whole procedure for both eyes need not take more than a few minutes and is therefore quite convenient.

One might argue that this test would not always work well, because it depended altogether on the faithfulness of the examinee, and a color-blind person might simply refuse an equation between two complementary colors. This objection has been raised even by *Mauthner* in the new edition of his *Farbenlehre*, 1894, p. 127. But it will not be difficult to catch such an individual by putting without his knowledge a piece of red glass in front of the color-tube, so that only red rays will enter the instrument. Under such conditions the two discs must appear alike to him in a certain position of the arc, so that he will be induced more easily to admit the equality of the color fields, when the red glass is taken away and replaced by common plane glass. But should he refuse to see two equal colors even with the red glass, then he would betray his attempt to deceive and be caught by his very denial.

In the construction of this color-tube a quartz-plate of 5 *mm* thickness was used, but it would be well to arrange the tube so that one or two 5 *mm* plates more might be dropped in, because then the colors are much less brilliant and saturated, and therefore on account of their whiteness better enabled to catch even the slightest cases of color-blindness. But in the writer's own experience the 5 *mm* plate has been always sufficient.

The advantages of such a method for those who possess a Javal and therefore have to spend only a few dollars for the color-tube, are the following:

1. The colors will never fade or change in any other way as long as white light is used for the test.
2. One need not cover one eye, as only one eye can see at a time.
3. The color-fields are large, and it requires no accommodative effort to view them.
4. It is possible to make the two different colors appear exactly alike, while in the worsteds one must be content with an approximation.[1]

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[1] Queen & Co. of Philadelphia will furnish these color-attachments.

A CASE ILLUSTRATING THE BAD EFFECTS WHICH THE ESTABLISHMENT OF MENSTRUATION MAY HAVE UPON THE COURSE OF INTERSTITIAL KERATITIS.

By JOHN DUNN, M.D., Richmond, Va.

On February 20, 1895, I was consulted by Mrs. M., in regard to the condition of her daughter's eyes. Such examination as could be made in the presence of the most marked photophobia I have as yet met with revealed double interstitial keratitis, of an exceptionally severe type. It had existed in the right eye four weeks, and in the left eye one week, when I first saw the patient. Miss M's facial appearance and notched teeth told the cause of the trouble. Mrs. M. remarked that, although her daughter was fourteen years of age, menstruation had not as yet been established. Miss M. was put upon the usual constitutional and local treatment. Her general condition rapidly improved, as was shown by her gaining much in weight. Improvement in the condition of the eyes was slow, but gradual. The photophobia, however, remained intense; so much so that on March 18th Miss M. was obliged, for comfort's sake, in the presence of either gas or daylight, to tie over her dark glasses a silk handkerchief folded several times. She went to bed the evening of March 18th, with her eyes showing this degree of photophobia. *When she awoke on March 19th the photophobia had entirely disappeared and with it practically all evidences of active inflammation of the corneæ. Menstruation had become established during the night.* As Miss M. lived in a distant city it was about a week after the disappearance of the photophobia before she was brought to my office. The condition of the eyes was then as follows: There were no traces of photophobia; indeed, the eyes could bear a light which would have been uncomfortable to a normal eye. There was, O. D., no redness of the conjunctiva along the corneal margin. The damage done to both corneæ, however, by the establishment of menstruation was beyond the hope of repair. Both corneæ were more or less cloudy; the left being more so than the right. Examined under a + 20 D sph. lens, the right cornea could be seen to contain numerous enlarged lymph channels and blood-vessels, the general direction of which was from periphery towards the centre (a condition Hirschberg and others have carefully described).

These lymph channels varied much in size and manner of distribution, some portions of the cornea showing large numbers of them, while others showed fewer. The left cornea was so milky (this milky appearance being in places in patches, as it were) that the normal corneal transparency no longer existed and the transmitted light was so little in amount that a strong plus lens failed to reveal the existence of the lymph channels so numerous in the right cornea. Vision: O. S., perception of light and existence of objects held between eye and light. All signs of active inflammation of the right eye had disappeared; not the slightest photophobia. The left eye still showed slight evidences of pericorneal irritation, but no photophobia. The establishment of the menstrual flow had, then, caused the interstitial corneal inflammation to cease abruptly, leaving the corneæ in the impaired condition to which the keratitis had brought them, and at the same time had removed the necessity for continuance of the inflammation until the corneæ had regained their normal transparency. The lymph channels seen in the left eye so plainly will in all likelihood remain visible under a strong plus lens as long as the patient lives, while there is little hope that the left eye can ever regain accurate vision. Time will modify somewhat the condition of the corneæ, but the changes now present must be considered as though they were of the nature of scar tissue, and time will modify them only as it does scar tissue non-specific in its origin. The character of the inflammation which renders possible the formation of these abnormal lymph channels in the corneal tissue and later their gradual diminution to final disappearance is different from any which we can induce by artificial means in a healthy eye; hence arises the difficulty of obtaining by treatment results such as we desire. A further lesson seems to be taught by the above case. In cases of interstitial keratitis it should be our object to use only such treatment as will improve the patient's general health; we should avoid any treatment having for its object the abrupt lessening of the duration of the inflammatory processes when once they have become fully established.

On June 20th, three months after the disappearance of the signs of active inflammation, the condition of the corneæ is as follows: There has been a slight clearing of the cornea, O. D.; the dilated lymph channels are, however, all through the corneal substance plainly visible under a + 16 D sph. lens. In O. S., the clearing up of cornea has been more marked, and this has resulted from the partial disappearance of the flaky opacities seen immediately after the disappearance of the photophobia. In this eye now, also, the dilated lymph vessels are visible in large numbers. The treatment during this time has been constitutional; frequent hot baths for the eyes. Electricity has been applied to the corneæ twice a week.

A CASE OF DERMOID CYST OF THE ORBIT.[1]

By B. L. MILLIKIN, M.D.,
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DERMOID cysts of the orbit, while not excessively rare, are sufficiently so to make a clinical report of each case interesting and important.

In 1880 Berlin (Gräfe and Sämisch, *Handbuch der gesammten Augenheilkunde*, vol. vi., p. 678) reported all the cases he could find at that time, amounting to seventy-three, of which twenty-three were doubtful genuine dermoid cysts. In looking up the literature since that time very few cases seem to be reported. The text-books on ophthalmology, with very few exceptions, give no extensive discussion upon this disease. One of the best articles I find upon the subject is by De Wecker and Landolt (*Traité Complet d'Ophtalmologie*, vol. iv., 1889, p. 821 *et seq.*), to which the reader is referred for an extensive discussion on the subject.

Berry, in his *Diseases of the Eye*, speaks of a single case having been operated upon in the Edinburgh Royal Infirmary, in 1867, but that he himself has not met with a case.

I find the following among the cases which have been reported since Berlin's article in 1880.

Hirschberg (*C. f. A.*, Band vii., p. 295) reports having removed a congenital lipomatous dermoid cyst in and behind the equatorial region of the eyeball.

Nicaise (*France Méd.*, No. 31) reports the removal of a dermoid cyst.

Dr. L. Webster Fox (*Med. News*, vol. xliv., 1884, p. 749) has reported four cases of cystic tumors, superficial in the tissues of the orbit, cured by incision and cauterization of the sac walls with silver nitrate.

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[1] Read before the Society of Medical Sciences, Cleveland, June 17, 1895.

J. L. Thompson (ARCH. OF OPHTH., vol. xii., 1883, p. 183) reports a cyst of considerable size, containing dark fluid, cured by electrolysis. It is very doubtful if this is a genuine dermoid.

Vignes (*Rec. d'Opht.*, Par., 1891, 3, 5, xiii., 409, 412) has reported a dermoid cyst at the internal portion of the orbit.

Licharewsky (*Wjestnik Ophth.*, 1887, No. 2, p. 129) removed a cyst the size of a pigeon's egg from the upper, outer portion of the orbit, with preservation of the globe.

Krönlein (*Beiträge zur klin. Chir.*, vol. iv., 1) reports the removal of a dermoid with two cysts, connecting through a fissure in the outer bony wall of the orbit, one cyst being in the orbit, and the other in the temporal fossa.

Polaillon (*Rec. d'Opht.*, June, 1886) reports a case of the left internal orbital region.

Dunn (*Am. Jour. of Ophth.*, vol. i., p. 39) has reported an interesting case of a girl of sixteen, with dermoid tumor, beginning at the inner angle of the eye, apparently above the lachrymal sac.

Pooley (*Ophth. Record*, vol. i., 1891, p. 181) removed a dermoid tumor by a long incision extending from the outer to the inner angle of the orbit, allowing the wound to heal by granulation, this being the second operation on this case.

Cornwell (ARCH. OF OPHTH., vol. xi., p. 338) has reported an interesting clinical case, likewise healing by granulation after a second operation.

J. Mitvalsky (ARCH. OF OPHTH., vol. xxii., 1892, p. 378) presents an extensive microscopical study of the subject of dermoid tumors, in and about the orbital region, in which he refers to four cases by other operators, and two cases which came under his own observation. The paper is based upon the study of fourteen cases of orbital and peri-orbital tumors, and the reader is referred to this article for a most exhaustive report upon the study of these growths.

Since 1892 I have found no cases reported in the literature at my command.

In these notes I have simply given orbital or intra-orbital cases, not referring to the numerous cases where the tumor is upon the orbital margin or in the peri-orbital region. All of these cases have been intra-orbital.

The position of predilection, according to Berlin, was found to be in 53 per cent. upon the nasal side of the orbit, while 24 per cent. were upon the outer portion. In 15 per cent. the situation is directly below and 4 per cent. directly above. In the cases reported since, the position has been in the majority of cases outside of the centre. My own case is, primarily, undoubtedly, in the latter location, that is outside of the median line of the orbit.

Case.—Frank R., æt. twenty-one, Bohemian, a large, muscular man, almost an athlete in build, came to my office on June 14, 1894, with the following history. Since thirteen or fourteen years of age he had noticed over the left eye an enlargement, which increased pretty rapidly until seventeen years of age. Since that time he does not think the size of the tumor has greatly augmented. At twelve years of age he remembers having been struck with a stone, somewhere above the eye, but does not remember just how long afterward the tumor of the eye began to develop, and from the subsequent history of the case the probabilities are that the blow had nothing to do with its development.

On examination I found the following condition present: O D v = $\frac{6}{6}$, O S v = $\frac{6}{9}$. The right eye is perfectly normal in every respect. Interior of both eyes normal. In the superior portion of the left orbital region there is a marked bulging of the tissues, protruding beyond the line of the orbital ridge, extending from the outer to the inner angle, slightly more marked within the median line. The eyeball is pushed forward, downward, and outward. The movements of the eyeball are apparently not restricted, and at no time has he noticed double vision. The upper eyelid droops slightly. Under the finger there is a large mass, quite hard in consistence, filling up the entire orbital space above the eyeball, apparently in the region of the lachrymal gland, and extending on either side to the angle of the orbit.

At the time I was inclined to believe the tumor to be connected with the lachrymal gland, but a positive diagnosis was not made. Removal of the growth was advised. This was inconvenient for the patient at the time, so its removal was postponed. No pain had ever been present, and no discomfort from the growth had ever been experienced by the patient, other than its cosmetic effect.

On Feb. 11, 1895, I again saw the patient. The condition at this time presented no appreciable variation, and apparently the size had not increased during the interval.

On Feb. 14, 1895, the patient being etherized, the following operation was performed: A long incision extending from the outer to the inner angle of the orbit, in a circular line, following the lower border of the eyebrow was made. Careful dissection was made through the skin and fascia down upon the tumor just at the upper margin of the orbit. The attempt was made to follow as close along the orbit as possible, in order to produce the least amount of disturbance of the tissues connected with the eyeball.

Upon reaching the tumor it was found that the contents were fluid, and the walls excessively thin and somewhat transparent. In endeavoring to dissect out the tumor, it was found impossible to remove it without rupture on account of the extreme thinness of the cyst wall. Rupture took place, and the contents escaped. This consisted of an albuminous-like fluid, probably more than half an ounce in amount, filled with apparently epithelial *débris*, and about the consistence of the white of egg. There was then complete collapse of the cyst and of course no certain dissection of the entire walls could be made. The cyst had extended entirely across the vault of the orbit, and deep down, almost to its apex. Just outside of the median line and close to the vault of the orbit, about the middle portion, antero-posteriorly, was found a large collection of hairs and epithelial *débris*. The hairs were many of them nearly an inch in length and brown in color, a shade or two lighter than the hair of the patient's head. Several forceps full of these hairs were extracted from this deep portion. Upon investigating in the same region with the finger I found a sharp spur of bone projecting from the vault into the cavity of the orbit, perhaps one quarter of an inch in length. The bony spur was about the size of an ordinary eye tooth. This seemed to have been the centre about which the growth had developed, as here the solid portions of the contents of the tumor were located. The walls of the cyst after rupture were deep red, or almost purplish in color, showing a high state of vascularity. As extensively as could be done the cyst wall was dissected away, the contents entirely cleared, and the tissues, where doubtful, were irritated thoroughly by raking with a burr. It was hoped that by thus raking the cyst wall inflammatory action might be developed sufficient to destroy it, and to permit it to become adherent to the neighboring tissues, without a tendency to redevelop.

In a growth so extensive, and with walls so thin, it was simply out of the question to dissect the cyst wall out in its entirety.

Thorough asepsis was carried out in all the steps of the operation, and the cavity left after the removal of the tumor was carefully washed out with a bichloride solution, 1 to 5000. The edges of the wound were then carefully approximated, and sutures introduced to hold them in the proper position. No drainage tube was employed.

There was profuse hemorrhage from the cyst and its walls during the different steps of the operation, but no vessels required to be tied. Hemostatics were used freely to staunch as much as possible the flow of blood. Oil protective and iodoform gauze were placed over the line of the incision, a gauze compress was made to press firmly the eyeball back into the orbital cavity, and all was kept in place by a firm roller bandage. The patient was put to bed and kept quiet for twenty-four hours. The next day he complained of the frontal portion of his head being "dead," evidently the supra-orbital nerve having been divided in the operation.

The wound healed perfectly by first intention, and the bandage and compress were removed on the fifth day, nothing being placed over it but a bit of adhesive plaster. The stitches were all removed on the sixth day, and the wound found to be entirely healed. At first there was difficulty in raising the eyelid, and there was great lack of feeling over the left frontal portion of the head. The eyeball was in good position, and its movements were entirely normal. There had been no affection whatever of his vision, in fact the sharpness of his vision had increased by the 7th of March, three weeks after the operation, and was at that time $\frac{6}{6}$. The only disturbance of the eyeball was a slight subconjunctival chemosis at the outer angle. Since the operation there has been a gradual improvement in the ability to raise the lid. When last seen he was able to open the eyelid fully one half.

There existed a peculiar and interesting condition of the bones about the orbit quite worth noting. The edge of the orbit above was very much thickened all along the upper margin, rounded, and with no sharp angle as in the opposite eye. This extended from the outer to the inner angle, and affects quite materially the entire shape of the orbit. The floor of the orbit in front seems to be somewhat depressed. This is interesting as showing the effect upon the development of the bones from the pressure upon them of the orbital growth. Other than a slight drooping of the lid there is now no disturbance, and the scar is simply a linear one following the line of the brow and scarcely detectable. Thus far there has been no indication of any return of the growth, but it is too soon to definitely decide so important a question.

I find that in many of the cases in which recurrence has taken place this has been noticed within a comparatively short period after the first operation. Many of the cases reported have suppurated, and of course the process of healing has been a very different one from that of primary intention. In not a few the eyeball, or vision, has been sacrificed.

The treatment of all cases of dermoid cysts should be operative. Whether or not an attempt at removal is the wisest course to pursue, must be determined by the nature of the growth itself in each individual case. If the diagnosis were positive, and if we were certain that no hair or other extraneous material were present, there could be no objection to the method of aspiration and injection with an irritating fluid, such as iodine, etc. Otherwise, undoubtedly, the best course is removal.

In small tumors with thick walls the removal of the entire cyst with its walls would not be particularly difficult, but with large tumors, with thin walls, the complete removal of the cyst with its walls I believe to be impossible, even in the most skilful hands. Under such circumstances the dissection of as much of the cyst wall as can be removed, and the thorough irritation of the rest by scraping or scarifying, would develop sufficient inflammation to produce adhesions of its sides and obliteration without suppuration. If union by first intention can be secured, the advantage of it must be apparent to every one. Not infrequently abscesses are the result of injections; they frequently produce very severe reaction and may be painful to the patient.

I cannot but believe that removal, as complete as possible, should be attempted in the vast majority of cases, as being a better surgical principle for the treatment of these conditions than any other.

TRAUMATIC PARALYSIS OF THE ABDUCENS NERVE.

By H. MOULTON, M.D., Ft. Smith, Ark.

THE two cases of traumatic paralysis of the abducens nerve reported by Zimmerman in these ARCHIVES, vol. xxiv., No. 2, with those previously reported by Purtscher and by Friedenwald, when added to the following case from my practice, constitute a round number of sixty cases recorded so far.

On May 20, 1894, a boy, aged eleven years, fell from a bicycle on to a brick pavement. Immediately afterwards a carriage wheel passed over the boy's head, the left side of the head being next to the pavement, the right side receiving the weight of the wheel. Thus the head was crushed by a directly transverse force, and I am told that it was perceptibly flattened. Blood escaped from both ears and nostrils. Consciousness was completely lost for ten days, gradually returning after that time. On the recovery of intelligence the patient complained of double vision. It was noticed that he was deaf on the right side, and that the right side of the face was immobile, and that the right eyelids had lost the power of voluntarily closing.

The case was brought to me on July 12th, when I saw it for the first time. There was complete paralysis of the right auditory nerve and of the right facial nerve. The diplopia was manifest when the patient looked straight in front, and increased on looking to the right. For the median position single vision was obtained with an 8° prism. Acuteness of vision and refraction were normal. There were no ophthalmoscopic signs. Intelligence and the general bodily functions were good. No visible marks of fracture were discoverable in any of the cranial bones. Six months after the first visit of the patient, the abducens had so far recovered that diplopia was only manifest on looking about 15° to the right; and at the present time, one year after the first visit, it is only manifest on looking *extremely* to the right. The facial paralysis has almost completely disappeared, but the acoustic paralysis remains complete.

This case was probably primary, though the abducens paralysis was not observed during unconsciousness, the symptom not being then looked for. From the direction of the force and the associated paralyses, the paralysis of the abducens, together with that of the other nerves involved, was probably produced by a longitudinal fracture of the petrous portion of the temporal bone.

DIPLOPIA OCCURRING AT THE PERIPHERY OF THE FIELD OF FIXATION.

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(*With 3 figures.*)

A PAPER, by Dr. A. Duane, read before the section of Ophthalmology, N. Y. Academy of Medicine, and published in the ARCHIVES OF OPHTHALMOLOGY, vol. xxiii., Nos. 1–2, entitled "Paralysis of the Superior Rectus," has suggested the advisability of studying the subject of the diplopia which is known to occur in *normal* eyes at the limit of the field of fixation, since his diagnosis of this affection, which he alone has found so common, is based upon diplopia in this portion of the field. The method pursued will be, first, to consider the reasons why diplopia appears; secondly, to study the behavior of the double images; and finally, to show its occurrence in practice and how the results compare with the observations of Dr. Duane.

1.—*The Cause of the Diplopia.*

For the purpose of carrying out the following experiment we adjust the head, rendered immovable by some device, in the primary position, in the centre of the arc of a large perimeter, so that the line joining the centres of rotation of the eyes is coincident with the cross diameter of the perimeter, and their middle points the same as shown in Fig. 1, where R and L represent the centres of rotation for each eye respectively and $o\ d\ o'\ e$ the arc of the perimeter. We then observe the greatest possible angle through which each eye can turn and still see clearly a small letter fixed on the slide. In this way we map out on the usual chart the limit of the excursion of each line of vision—that is, the field of monocular fixation, *r r* and *l l*, Fig. 2. It is evident, however, that the true angular deviation is not obtained in this experiment, since by the modification of the usual arrangement each eye lies, not in the centre of the arc of the perimeter but is removed one half the distance between the centres of rotation (30–35 *mm*).

Further, in most portions of the field of fixation it is possible to see the object, although indistinctly, if it is moved

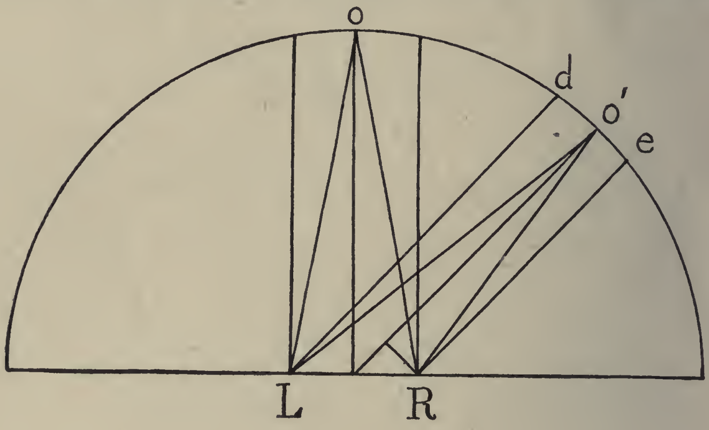

Fig. 1

beyond the field of fixation, and there can be traced, beyond its limit, a field of vision *R R* and *L L*, the boundaries of which are formed by the intervention of the brow, nose, and cheek on the upper, inner, and lower sides. The two fields for each eye in my own case are shown in Fig. 2, obtained by means of after-images of a bright light at the macula, which follow exactly the motion of the line of vision. It will be seen that beyond the point 45° to the right side, the line of vision of the left eye cannot turn and the limit for the right eye is 55°. As an object passes beyond the limit of motion for the left eye its image will fall outside the macula although the right eye can still fix upon it, hence it is obvious that *diplopia* must occur and that *crossed.* There are three conditions under which diplopia might not occur at the near point in this part of the field. Either the internus might exceed the externus in power by enough to equal the difference as shown (about 10°), or the nose must cut off the field on the inner side before the limit of fixation is reached, or there must be an additional stimulus to the internus accompanying the effort at convergence.

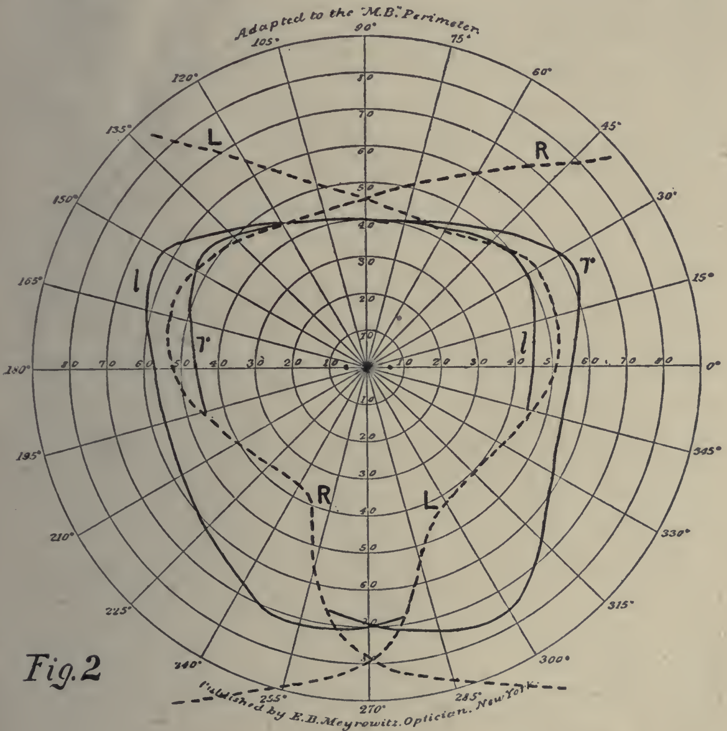

Fig. 2

1. In regard to the comparative extent of motion for each eye Helmholtz gives 50° as the greatest possible extent of motion, both inwards and outwards. This is about the case in my own eyes, outward motion, however, being a little stronger (2°) than inward, which is contrary to the rule, for, according to the average of eight authorities given by Hering, the inward excursion of each eye is 45°—the out-

ward 42°. In normal eyes examined by me I found the angular deviation nearly equal for inward and outward motion with a difference of not more than 4°.

2. In reference to the intervention of the nose it is seen in Fig. 2 that in my own case there is a space of about 6° where diplopia must occur at the near point. As one would suppose, this space varies in individual cases, but out of 14 cases examined in reference to its existence I found only one case in which the line of vision could follow an object in the motion inwards until it was cut off by the bridge of the nose. As determined on a perimeter of 30 *cm* radius, this space where unconquerable diplopia occurs, varies from 0° to 12°.

3. One would not suppose that, after the eye was turned inwards to its greatest extent in distant vision, still further adduction could be brought about by the effort of convergence, nor do we find that this is possible, indeed quite the contrary condition is asserted by Hering, namely, that the binocular field of fixation is less than the part which the two monocular fields have in common—that is, that the space enclosed by the lines *R*, *L*, *l*, *r*, Fig. 2, does not represent the binocular field of fixation. This was by no means true in the cases observed by me, but would account for diplopia irrespective of the conditions referred to above.

Another method of demonstrating the limit of motion for the eyes is, with the left eye closed and with the other fixed on a distant object lying a little above the horizontal plane, to turn the head on a vertical axis to the left away from the object with the eye still fixed upon it. As the eye reaches its limit of motion, the object of fixation and the whole field will seem to revolve in the opposite direction to the motion of the head. This happens also when the other eye is turned inwards, showing that the limit of motion is reached before the nose intervenes.

Having seen the causes for diplopia in extreme lateral motion, we pass to consider the field of fixation when the eyes are turned directly *upwards*. At this point it is necessary to state a fundamental principle which underlies the phenomena of the motility of the eye, namely, that being commonly accustomed to regard objects which are near at hand in looking down, distant objects in looking up, we find convergence more difficult in the upper part of the field of fixation. This convergence, however, I find is generally possible in extreme upward motion, if the object is not too near the eyes. Thus uncontrollable diplopia does not necessarily occur above at the near point, except beyond the limits of the field of fixation when, according to the law of corresponding points on the retina, an object will appear double if it is outside the total horopter. Hence, as in this case, the horopter is a line inclined with its upper end toward the observer; a vertical line, if its lower end be fixed, will appear double with the two images divergent. This is difficult to observe. It can readily be seen in view of the difficulty, or in some cases impossibility, of fixation at the near point above, that divergence may easily occur. This is actually the case, and as a consequence lateral diplopia, obviously crossed, is of common occurrence in the upper field. At an infinite distance diplopia does not occur above, as a rule, because the lines of vision are parallel and seem to remain so when the object of fixation passes beyond the limits of the field, and because the horopter is then an infinitely distant perpendicular plane; either crossed or homonymous diplopia is possible, however, in individual cases, the tendency being toward the former.

In the *lower* part of the field of fixation diplopia is not found at the near point, but homonymous diplopia occurs in some cases at a distance, from the tendency to convergence below, in accordance with the principle enunciated above. In the few cases in which I have tried this, I get homonymous diplopia at a distance in some, but only at the extreme lower limit, although the tendency is always present. In others, and in my own case, the diplopia is conquerable even to the very limit of the field.

In the *upper lateral* parts of the field, according to the diagram, unconquerable diplopia does not occur. It must be observed, however, that by raising the eyebrows, as is usually done in extreme upward motion, the field of vision is considerably extended beyond the field of fixation, and under those conditions I get unconquerable diplopia. Practically it is with difficulty that an object can be fixed at the near point, at the limit of this portion of the field, on account of the strain in maintaining convergence with upward and lateral deviation, and diplopia readily occurs, in many cases unconquerable.

In the *lower lateral* parts of the field diplopia cannot occur on account of the fact that the nose cuts off one eye.

It must be said in reference to the foregoing observations that variations in almost every particular occur in individuals whose muscular apparatus may be said to be normal; the experiments are in parts of the field rarely used and with the muscles under strain; further, the muscular power varies from day to day or even during the examination. Patients will also often make a momentary effort which they cannot sustain, in many of the tests. Finally slight variations in the laws of motion of the eye are known to occur at the extreme periphery of the field.

2.—*The Behavior of the Diplopia.*

If, with the head fixed and the eyes directed upwards and to one side, to a point on the opposite wall, we bring a pencil with its point at a distance of 30 *cm*, in the plane of fixation (which is a plane determined by the centre of rotation of each eye and the fixation point), it will appear double, the diplopia being crossed. The point of the pencil in each image will be at the level of a horizontal line on the wall containing the fixation point, such as the cornice, and this experiment, repeated with the greatest care in reference to the primary position and immobility of the head, shows that the double images of an object at the near point formed during divergence in a given elevation of the plane of fixation occur in this plane of fixation and are projected on to the horizontal line in which this plane cuts the wall. If now we turn our attention to the relative heights of the two images, a fact appears to which I wish to call especial attention, namely, *the inner image seems the higher* Horizontal lines on the opposite wall we judge to be horizontal, hence it is evident that the images are not referred to the plane of the wall. Each image is, in fact, localized in space at the distance of the real object and on a sphere at right angles to the line bisecting the angle made by the two lines of fixation, hence passing through the middle point of the imaginary line joining the centres of rotation (which point is the centre of the sphere) and through the fixation point. If we push a pin through a card so that it projects perpendicularly from the other side, then with the head level and the eyes in the oblique position (upwards and to the right) hold the card in such a way that with one closed the image of the pin is exactly in the line of vision

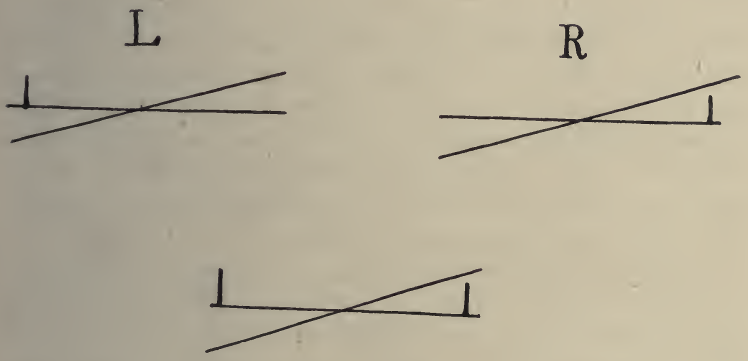

Fig. 3

of the other eye, and appears as a point, by rotating the card about the pin as an axis until its upper edge is coincident with a horizontal line on the wall, that is, until it lies in the raised plane of fixation, we have an inexact representation of the surface to which the double images are referred, and it is apparent from the evident inclination of the upper edge of the card, why double images in the plane of fixation seem at different levels in reference to the plane of the horizon. When the other eye is open the double images of the edge of the card in divergence are in the same straight line. Fig. 3 shows the formation of the images on the retina, as seen from behind, when the eyes are directed upward and to the right. The oblique line represents the position of the retinal horizontal due to the wheel-rotation (Helmholtz) as shown by an after-image; the horizontal line shows the line in which the plane of fixation cuts the retina, and it is along this line that the double images always occur. The lower figure gives the two retinæ superimposed.

The vertical diplopia occurring as described increases with the deviation of the eyes from the median plane (where the images appear at the same level) and also with the elevation of the plane of fixation. The amount of vertical diplopia can be calculated from the formula given by Helmholtz for determining the wheel-rotation in a given position of the eyes.

In regard to the distance between the images, this will vary (1) with the amount of relaxation, being greatest when the lines of vision are parallel; also (2) with the distance of the object from the eye, and finally (3) by the drawing together with lateral motion of the parallel lines of vision, as shown in Fig. 1. The image belonging to the nearer eye appears the larger, the other is more distinctly seen because it is easier to accommodate for this.

In order to understand the tilting of the double images of a straight line or a candle, it is necessary to repeat the experiment of Meissner, by suspending a black thread in a vertical position before the eyes and by voluntary divergence, or if one has not learned to accomplish this, by fixing a point just behind the thread, causing the thread to appear double. The two images of the thread appear to *diverge* above, but by inclining the thread with its upper end away from the observer, in the median plane, at the proper angle, they will appear parallel—that is, will be in the horopteric line. The tilting is greatest when the two images are nearest together. This phenomenon is due to a rotation of the eye outward by convergence, which causes divergence of the apparent vertical meridians of the retina, and also to the divergence of the apparent vertical meridians, in parallel sight, due to an incongruity of the retinæ, which is normal in varying degrees. The divergence of the upper ends of the two images also takes place in the diplopia occurring in extreme lateral motion. In looking upwards the images caused by slight divergence, as described above, in order to appear parallel must be inclined away from the perpendicular to the plane of fixation thus raised, but by no means as far as a vertical position. If however the thread is carried through a greater angle into a vertical position, then the double images seem to *converge* above, because now the upper end has passed to the other side of the horopteric line, and the same is true in lateral positions in the raised plane of fixation. Patients will rarely observe the tilting unless attention is called to it. The tilting as described above I have been able to confirm with more or less exactness in a number of cases examined with this point in view.

3.—*Occurrence in Practice.*

Having shown the causes for the existence of diplopia and the behavior of the double images, we turn to inquire whether it actually occurs in practice, and if so, what the patients tell us in reference to the images. We are at once confronted with a difficulty, in that the vast majority of individuals will not recognize double images even when the conditions which produce them are present. For instance, if a patient is told to gaze at a distant object, his finger held before the eyes will not ordinarily appear double, although almost every one can be taught to see the two images. Hence out of 56 individuals examined, during two months' time, for diplopia at the near point, 11 saw double at once in some portions of the periphery of the field; 7 in upward, lateral, and oblique positions; 3 in lateral and oblique, and 1 in lateral positions only. Out of 45 remaining, 41 saw double if attention was called to the two images when looking beyond the object, and diplopia of the character described above was always present at the periphery. Four could not be made to see double under any circumstances. The point of especial interest is that of the 52 cases who saw double, 49 said that in the oblique position the inner image seemed higher than the outer. In two cases they seemed at the same height, being probably referred to the plane of the wall. A few declared that there was a difference in the vertical diplopia on the two sides. This was due either to a slight tilting of the head, which is involuntary and difficult to prevent in clinical observations, and at once affects the relative heights, or to the presence of hypophoria. Whether due to the former cause or not, it is easily determined by the fact that diplopia will not be present at a distance, whereas if hypophoria is present, and the variation is due to an actual difference in the elevation of the two lines of fixation while the centres of rotation are in a horizontal line then diplopia must occur for distance, at the periphery. Diplopia at a distance at the periphery of the field is of common occurrence, and, as would be expected, insufficiencies of the interni, as a rule, show crossed diplopia of the externi, homonymous. Vertical diplopia with or without lateral diplopia occurs above in many cases of hypophoria. Unconquerable diplopia I find to be more common above at the near point in insufficiencies of the interni. Dr. Duane in the paper referred to above, makes the statement (p. 74) that "the characteristic feature is contained in the expression Eu & r D L > > [which interpreted means: eyes up and right, vertical diplopia increasing with the outer image the lower], which alone is sufficient to make out the presence of this condition" paralysis of the right superior rectus, also stating that D X < < [crossed lateral diplopia, diminishing] is present. Now this is exactly the behavior of the diplopia which occurs in *normal* eyes spontaneously or by voluntary divergence, as I have attempted to show. Hence it does not seem necessary to infer "congenital paralysis of the superior rectus" in order to explain the phenomena which he has recorded in the fifteen cases appended to his paper.

A NEW METHOD OF TYING THE SUTURES IN ADVANCEMENT OF THE OCULAR MUSCLES.

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(*With four figures.*)

TO bring this before you I will describe in detail the advancement operation at present practised by me, although not claiming any portion of it as original except the manner of fastening and tying the sutures. This method is equally as serviceable in advancements for heterophoria as in true squint.

The first step in advancing the muscle in, for instance, convergent strabismus with a turning in of say 5 *mm*, would be a tenotomy of the internal rectus muscle.

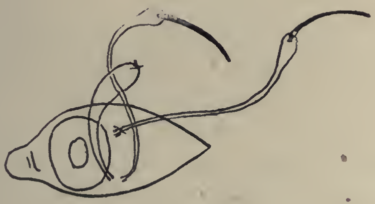

Fig. 1.

Second step. Fastening sutures.—Thread a full curved needle with No. 3 iron-dyed silk, bringing ends of thread together and tying in a small hard knot, or both ends of the thread may be passed through the eye of the needle at the same time, leaving the end of the suture in the form of a loop instead of a knot. The needle is now passed through the conjunctiva, taking a good bite into the sclera close to the cornea, as indicated in drawing No. 1.

After the thread is pulled about half way through, the needle is passed through between the threads on the other side of its entrance into the sclera and then drawn home thus affording a firm point of fixation. A similar suture is fixed in the same manner upon the opposite side of the cornea.

Third step.—The belly of the muscle to be advanced is now picked up with a pair of mouse-tooth forceps through the conjunctiva; with a pair of scissors the conjunctiva is snipped through both above and below the muscle, at the same time freeing any submuscular attachments from the globe. A tenotomy hook is now passed underneath the muscle, and the conjunctiva and submuscular tissue cut horizontally towards the cornea and then towards the external canthus and the belly of the muscle thoroughly freed from the globe. (See drawing No. 2.)

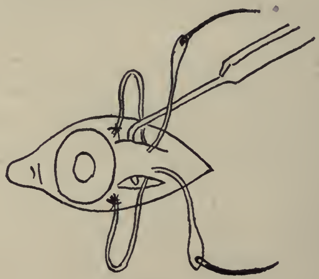

FIG. 2.

The sutures are now passed through the belly of the muscle from below upwards as far back as is believed necessary, and the sutures pulled about half way home. The muscle is still being held up with the tenotomy hook, and is now cut off just in front of the entrance of the sutures. The hook being of no further service, is supplanted by a pair of fixation forceps, with which the short piece of muscle attached to the globe is grasped and cleanly dissected from the globe. (See drawing No. 3. The portion of muscle removed is shaded.)

The sutures are now pulled home, and both grasped between thumb and finger, while the globe is fixed with forceps on nasal side of cornea and turned outward, while the muscle is advanced to the desired position, or until the eye is in its proper position. I do not desire an over-effect.

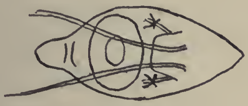

FIG. 3.

Fourth step.—The sutures are now tied in a surgeon's knot over the muscle, as indicated in drawing No. 4.

The advantages of this suture over any other method I have tried are that, as the eye is drawn into position, both sutures can be controlled with one hand, leaving the other free to turn the globe in the desired direction with forceps. There is no rotation of the globe upon its anterior-posterior axis, as the tension upon each suture is the same, and this equal tension is maintained throughout the time of healing. The sutures are easily removed by snipping the threads anywhere between their scleral and muscular attachments, grasping the knots with forceps and lifting them out. I find it is not necessary to leave them in longer than four days. The operation described can be performed under cocaine subconjunctivally injected. There is no deformity at the site of operation, and within a month's time it is difficult to see that an operation has been performed.

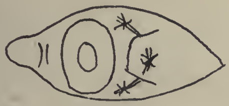

FIG. 4.

A CLINICAL STUDY OF ONE HUNDRED AND SIXTY-SEVEN CASES OF GLAUCOMA SIMPLEX.

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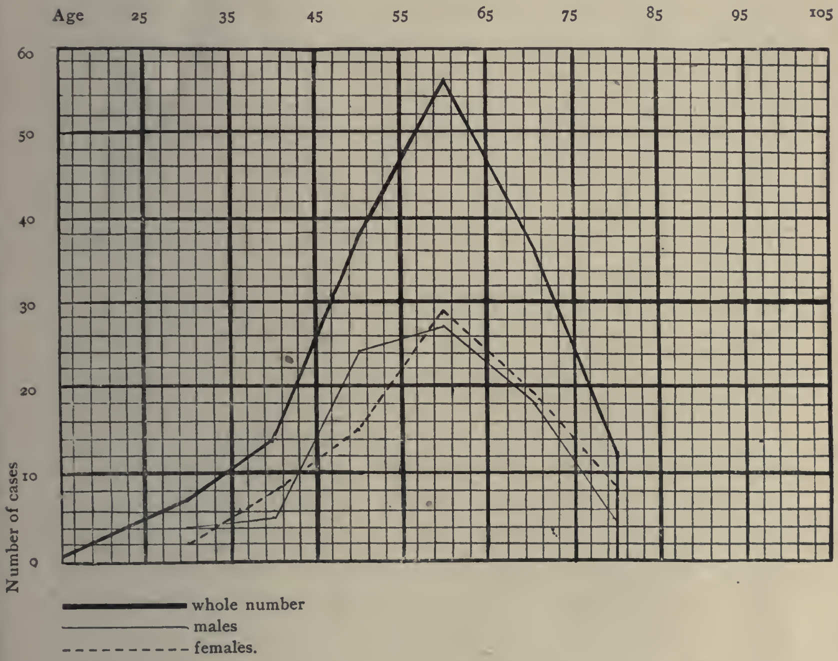
THESE cases were observed in the services of Doctors William F. Norris and Charles A. Oliver at the Wills Eye Hospital, Philadelphia. The authors wish to thank these gentlemen for permission to publish these findings, and to express their appreciation of the careful and thorough clinical methods which have made such a study possible, and which have added so much to the value of the article.

It has ever been the custom in these clinics to note the full description of each eye, including the ophthalmoscopic appearances, in every patient who was admitted to the clinic, so that the percentages which have been deduced from these findings must be more nearly accurate than those obtained in clinics where the practice has been to employ the ophthalmoscope only where it seemed particularly indicated. In view of the fact of the large number of patients who came complaining of affections of the external coats of the eye or of the adnexa, but in whom distinct glaucomatous symptoms were discovered, many incipient cases would have been overlooked had not the routine practice been rigidly adhered to in every instance.

The 167 cases occurred among 22,680 patients, or .736 %.

Men were found to be slightly more liable than women, 85 of the 167 cases occurring in males and but 82 in women.

Past middle life exhibited the best soil for the growth of the disease, although senility and extreme youth were not exempt. The extremes noted were 13 and 96 years. The next oldest cases, however, did not occur until between the ages of 75 and 85, the next youngest until between the ages of 25 and 35. But these extremes of age were exceptional, and the disease showed a marked preference to occur between the ages of 55 and 65 years, although the average of the total number affected fell within the preceding decade, being 52½ years. Of the total number, 1 occurred at 13 years; 6 between 25 and 35 years (2 of these at the age of 26); 13 between 35 and 45; 39 between 45 and 55; 56 between 55 and 65; 37 between 65 and 75; 13 between 75 and 85; none between 85 and 95, and 1 at 96. In one instance the age was not given. These figures are quite in



harmony with the ocular changes which, occurring as a part of or complicating the disease, suggest its senile character. The accompanying table, arranged after the form of Priestly Smith's, shows very graphically the relationship existing between the age and the sex in the production of the disease. It becomes at once apparent that at the age of 50, males are much more prone to the disease than women. This is quite at variance with what one would expect, as the climacteric, by its interference with the general circulatory system, would naturally be supposed to offer favorable conditions to the development of glaucoma. Thinking that these figures might be misleading, owing to a possible predominance of male patients at the clinics, the authors made a careful count of the number of males and females which comprised the total number from which the cases were drawn, and they found that of 22,680 cases, 12,089 were females, whilst but 10,591 were males. This proves conclusively the greater liability of males, for, in spite of their smaller number, they were affected in a higher proportion. After the sixtieth year both sexes seem to be almost equally liable, females slightly more so than men.

The authors wish to call particular attention to the value of this table, for it represents statistics based upon observations which they deem to be more complete than any which have been hitherto made. Priestly Smith's table is founded upon the observation of one thousand cases collected from different sources without mention being made of the proportion which they represented of the total number of cases from which they were drawn, nor of the sexual composition of the clinics in which they occurred.

Much has been said from time to time in regard to *systemic diseases acting as predisposing agents to glaucoma.* The history of previous systemic disease which is elicited in an ophthalmological clinic is unfortunately too often but scanty and unsatisfactory. The intelligence of such patients is often at fault, but more often there seems to be a reluctance upon the part of the patient to depart for one moment from the recital of his ocular symptoms to recall a general disease from which he may have suffered years before, and which to him bears no possible connection with his present local affliction. From the findings, however, it would appear that regard must be paid to two diseases in the production of this form of ocular disease—rheumatism and influenza. Whilst the influence of the latter was traced as a causal factor in but a small number of cases (7), nevertheless the relationship was so close in these that it must be given due importance. Influenza is a disease which has manifested itself with unusual severity during the past decade, and the authors are of the opinion that the increase in the frequency of glaucoma simplex, which was found to occur in the later years was due to the deleterious action of this affection. Twenty-one of the cases were subject to distinct attacks of rheumatism. When the age of the subjects is considered, and the exposure to which most of them as laborers of different kinds had been subjected, perhaps this proportion would not be higher than that found among those of the same age and in the same walks of life who exhibited no traces of glaucoma; nevertheless the proportion of cases in which rheumatism existed is large, and there seems to be good evidence to support the opinion that the arthritic variety of the disease especially predisposes towards the glaucomatous state. Typhoid fever was thought by the patients to have been a predisposing cause in 3 instances, and intermittent fever in 3, whilst dysentery, syphilis, epilepsy, dyspepsia, and the menopause were each held as accountable for the causation of the disease in 1 instance. All of these affections tend to lower the vitality, and it is probable that with the predisposition towards the disease already existing locally, it was precipitated by the impaired general condition which they wrought. At the same time with the ocular disease, in addition to the foregoing, which were supposed to have exerted a causative agency, there existed in a sufficient degree to have excited the comment of the patient cardiac disturbances in 5 cases, uterine disease in 2, and ozæna, apoplexy, and hemorrhoids in 1 instance. Fifteen claimed to be in good health, 9 were addicted to the use of tobacco, and 3 were intemperate in the use of alcohol.

Simple glaucoma being defined as a progressive affection not usually associated with any inflammatory signs or subjective sensations, it is of great interest to note *the causes which led those suffering from it to a consultation for* their ocular affection. In our 167 cases, 39 sought treatment on account of failing sight, 19 on account of presbyopia, 19 on account of ocular pain, 5 on account of headache, and 3 on account of asthenopia; whilst the disease was accidentally discovered in a number of instances in individuals who sought relief from external affections, such as foreign bodies, catarrhal conjunctivitis, etc. As just stated, failing sight was the cause which most frequently led the patient to seek treatment.

Glaucoma is essentially a disease which is attended by loss in the visual acuity, but the absence of this symptom does not preclude the possibility of the glaucomatous process, for in 36 eyes in which there were other well marked symptoms of the disease, the central visual acuity was normal. It may be seen by this, and it is generally acknowledged, that *the degree of central visual acuity* is not a safe guide to the extent to which the glaucomatous process may have advanced, and when reliable information is desired of the visual condition recourse is usually had to a study of the peripheral vision as revealed by the perimeter.

Of the 167 cases, *failing sight was noted in both eyes* in 65 instances, whilst in 13 cases the defect was noted in but one eye. The disease is almost without exception binocular, and had these 13 cases delayed seeking advice somewhat longer they would undoubtedly have had to complain of loss of vision in both eyes. It was but rare that the sight of each eye became simultaneously affected, for it was found that after the loss of the visual acuity had first manifested itself in one, an average period of twenty months elapsed before it showed itself in the fellow eye. Even after the patient had become conscious of the fact that his sight had commenced to fail, the loss was so gradual and produced so little annoyance that it was found that an average of 3¼ years were permitted to elapse before it was deemed necessary to seek to improve it. The vision of the left eye was more affected in 66 instances, the right in 50, while both eyes were affected in a similar degree in 40 cases. Although the patients' statements in regard to the time elapsing after the appearance of failing sight before total blindness ensued or vision was reduced to light perception, must be given a great deal of latitude, the average time necessary to accomplish this unfortunate result was 2 years 5½ months. The following figures are of interest as showing the different stages of the disease existing in the eyes at the same time.

a. In 36 instances both eyes possessed full visual acuity, $\frac{5}{5}$ or $\frac{5}{7\frac{1}{2}}$.

b. In 6 instances one eye possessed full visual acuity, and the other was blind.

c. In 107 instances vision was reduced more than one half in both eyes.

d. In 62 instances the difference in visual acuity in both eyes was more than one half.

e. In 25 eyes vision was reduced to l. p., and in 14 it was totally abolished.

f. In 32 of the eyes with full visual acuity, there were distinct glaucomatous excavations.

After loss of sight *headache* was the most prominent symptom, 44 patients stating that they suffered from it. This symptom did not affect all similarly; 11 described it as being temporal in character, 14 as frontal, 7 as occipital, while in 3 instances it took the form of a hemicrania. Eight asserted positively that they had never had any pain in the head.

Ocular pain was present in 18 cases, whilst in 12 instances, where this symptom was suspected by the surgeon, the patient denied ever having had such a sensation.

Halo was noted in 8, and muscæ in 7 cases.

Glaucoma simplex is regarded as being a disease which runs its course without usually exhibiting any inflammatory signs. There are many cases, however, in which *irritation symptoms* manifest themselves, and this usually happens in the later stages of the disease. Fifteen of our cases were noted as having tarsal and ciliary congestion, but in several instances this was caused by accidental catarrhal disease of the conjunctiva. The vast majority were noted as being quiet, and, beyond the external manifestations significant of the pressure within the globe, there was nothing to denote an inflammatory process.

The *pressure symptoms* were as follows: The anterior scleral vessels were noted as being engorged in 28 instances. The cornea was variously described as "steamy" and "needle-stuck" in 58 instances. This membrane was anæsthetic in 22 cases, and in 10 instances it was irregular and maculated. There were abnormalities in this membrane in 26.94 per cent. Probably on account of the advanced age at which the majority of the cases presented themselves, arcus senilis was present in 60 instances.

Study was made of the *anterior chamber* in those cases where the presence and the character of the excavation was noted, and it was found that—

In 64 eyes the a. c. was shallowed and the discs excavated.

In 10 eyes the a. c. were shallowed and the discs not excavated.

In 3 eyes the discs were excavated, but the a. c. were normal.

In the 10 cases in which no excavations were noted, there were pathological excavations in the fellow eye, giving rise to the thought that these 10 eyes were in an earlier stage of the disease, as evidenced by the shallowed chamber, and that excavations would appear later. In the last 3 instances, it is only fair to suppose that the angles of the anterior chambers were blocked, although in a manner which was imperceptible to the clinician. Except in 3 cases, where the chambers were unaffected, increased tension was always associated with shallowed chambers. But the converse did not hold, for in 15 cases where the chambers were markedly narrowed, the tension was noted as being normal.

The pupils were 2½ *mm* in size in 75 instances. In 62 of these the irides responded freely to light and convergence, whilst in 13 they were sluggish to these stimuli.

The pupils were 3 *mm* in size in 62 instances. In 45 of these they responded equally and freely to light and convergence, whilst in 15 they were sluggish to these stimuli, and, in 2 instances, non-responsive.

The pupils were 4 *mm* in size in 38 instances. In all of these they responded equally and freely to light and convergence. In 17 they were sluggish, and in 10 non-responsive.

The pupils were dilated ad max. in 19 instances, in 5 of which they were sluggish, and in 14 non-responsive.

In regard to *the relationship existing between the size of the pupil, the presence or absence of an excavation, and the state of the tension*, a glance at the following table will show that neither increased tension nor the presence of an excavation was constantly associated with an enlarged pupil, although, in the vast majority of the cases—119 eyes (35.62 %)—where there were excavations, there were also enlarged pupils.

Table.

Pupils 2½ *mm*, T n., glaucomatous excavations = 15 cases.

Pupils 2⅓ *mm*, T +, glaucomatous excavations = 12 cases.

Pupils 3 *mm* or more, T n., glaucomatous excavations = 20 cases.

Pupils 3 *mm* or more, T +, glaucomatous excavations = 42 cases.

The media were noted as being clear in 47 instances. In addition to the 58 cases of corneal opacity, there were lenticular opacities in 95 instances. The nucleus of the lens was hazy in 36 cases, the cortex in 45, whilst in 14 the opacities were marked enough to have received the designation of cataract. Vitreous opacities were observed in 6 instances.

The Nerve.—Every nerve the seat of a glaucomatous excavation presented an abnormal tint. In 64 cases it was dirty red-gray, whilst in 41 instances it was described as gray. In 7 cases it had a greenish hue, and in 1 a bluish. In 2 it appeared waxy. It was hazy to the nasal side in 10 instances, and hazy all around in 2. In 5 instances there was a low-grade papillitis. The lymph sheaths were full in 6.

Excavations.—These were of all varieties, of varying degrees of depth and extent. Sixty-five were complete, 7 were undermined, 9 shallow, involving the entire surface.

103 were deepest out.	5 were deepest down and in.
35 were deepest down and out.	5 were deepest down.
7 were deepest up and out.	2 were deepest in.

The average depth of the excavation was 3.37 D.

Table.

Total number of eyes with pathological excavations possessing normal visual acuity = 32.

Total number of eyes with pathological excavations possessing normal tension = 72.

Total number of eyes with pathological excavations possessing increased tension = 91.

Total number of eyes with pathological excavations with contracted fields = 143.

Total number of eyes with pathological excavations with normal fields = 40.

In only 9 eyes with cut fields were there no glaucomatous excavations. In only 4 eyes with tension increased were there no glaucomatous excavation.

Glaucomatous excavations in one eye = 30.

Glaucomatous excavations in both eyes = 101.

Cases in which there was a probable excavation, but the haziness in the media prevented a view of the fundus = 21.

Significant and constant though the changes in the structure of the nerve head itself may be, those occurring in the scleral ring are no less worthy of mention.

Scleral Ring.—A broad scleral ring all around occurred in 147 cases; 33 times in beginning cases; 56 times in excavations to the temporal side; 40 times in complete excavations; 7 times in large shallow excavations, and 11 times in physiological excavations.

A broad scleral ring *out* occurred in 23 cases.

A broad scleral ring *in* occurred in 3 cases.

A broad scleral ring *up and in* occurred in 3 cases.

A broad scleral ring *down and out* occurred in 7 cases.

A broad scleral ring *in and out* occurred in 11 cases.

A broad scleral ring *down and in* occurred in 6 cases.

A broad scleral ring *up and out* occurred in 1 case (physiological excavation).

A faint scleral ring all around occurred in 3 cases.

Marked halo occurred 31 times. Choroidal rings and conuses showed a tendency to become absorbed to the temporal side, and more especially down and out.

The Visual Field.—A careful study was made of the visual field in 215 eyes, and a painstaking comparison of these fields with a view of determining the extent of the average field, which was instituted, resulted as follows:

The form field was contracted:

To 34.83° to the nasal side.
To 50.10° to the temporal side.
To 31.75° above.
To 43.71° below.

The contraction for red was:

To 20.53° to the nasal side.
To 31.75° to the temporal side.
To 19.35° above.
To 24.52° below.

Seventeen cases where the form field was abolished and the *candle field* was alone obtainable, showed an average contraction for this source of illumination to be—

To 26.27° to the nasal side.
To 33.81° to the temporal side.
To 34.81° above.
To 40.45° below.

In 10 of these cases, in addition to the glaucomatous process, the diminution in the field was occasioned by opacities in the media. These figures go to show that in the majority of cases the reduction in the peripheral vision took the form of a concentric contraction of the field throughout its entire extent. They show further that the disturbance in the perception of color goes hand in hand with that for form. In 32 cases, however, in which there were glaucomatous excavations the color field was found to be contracted proportionately more than that for form, and in 6 of the latter there were distinct cuts in both form and color fields.

In 44 cases (20.46 per cent.) there were distinct cuts, *i. e.*, entering angles in the field. These defects occurred in all portions of the field and did not show any predilection for any sector. In 19 of these cases, where there was concentric limitation to 20° to the nasal side, there was full visual acuity. Scotomata were noted as occurring in but 4 instances.

The extent of the visual field and the presence or absence of an excavation.—It was possible in 121 eyes to make a comparative study of *the tension*, *the vision*, and *the extent of the visual field* and *the presence or absence of an excavation.* The results obtained are as follows:

T n., good vision,[1] good field,[2] excavation present = 3.

T n., good vision, contracted field,[3] excavation present = 6.

T n., poor vision, good field, excavation present = 14.

T n., poor vision, contracted field, excavation present = 35.

T n., poor vision, contracted field, no excavation present = 5.

T +, good vision, good field, excavation present = 2.

T +, good vision, contracted field, excavation present = 6.

T +, poor vision, good field, excavation present = 10.

T +, poor vision, good field, no excavation present = 1.

T +, poor vision, poor field, excavation present = 37.

T +, poor vision, poor field, no excavation present = 2.

It will be seen at once by these figures that an excavation existed in all but 8 of these 121 cases. They also show how the diminution in vision, the contraction of the field, and the presence of the excavation go hand in hand. The influence of increased tension does not appear to have played so important a part, as it was noted as being normal in 53 instances, whilst there was full visual acuity in only 17 instances. In order to gain an idea as to which factor appears the more often a count was made of the total number of *ex-*

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[1] Vision = 5–5 or 5–7 1–2.

[2] 55° nasal for form.

[3] Contraction to at least 30° nasal for form.

cavations which had been noted as appearing in the 334 eyes. Two hundred and seventy-two excavations were found described and of this number 245 were glaucomatous.

To these there must be added 21 eyes in which an ophthalmoscopic examination was impossible on account of haziness of the media, but where there was a probable glaucomatous excavation as evidenced by the presence of the other three cardinal symptoms *i. e.*, poor vision, contracted field, and increased tension.

In like manner cognizance was taken of the state of the *tension.* This had been noted in 199 eyes and in 109 instances it was increased. The *fields* were found to have been studied in 215 cases and in 96 instances they were contracted. To these must be added 44 blind eyes and eyes in which vision was reduced to l. p.

Table.

Eyes containing excavations,	272.
Eyes exhibiting increased tension,	109.
Eyes with contracted fields,	240.

A spontaneous venous pulse occurred in 22 cases and an *arterial* in 20. Of these latter, the pulse was induced by slight pressure on the globe in 6 instances. The veins were noted as being tortuous 12 times and the arteries 8. It is probable that anomalies in the vascular distribution occurred in a greater number of cases, but escaped description, varying degrees of tortuosity being supposed to exist in all cases where the pathological process was marked enough to impede the vascular supply. In about 20 per cent. of the cases the choroid was described as being congested, while evidences of retinal opacity were sufficient to deserve mention in a few instances.

Refraction.

According to many observers *hypermetropia* predisposes toward glaucoma, the shape of the globe in this form of ametropia and the peculiarities in the accommodative effort which accompanies it, being held responsible for the causation of the disease. In simple glaucoma, however, the character of the ametropia appears to have no relationship whatever in inducing this pathological state. It is true that the great majority of the 167 cases were hypermetropic, that form of ametropia being recorded as occurring in 140 eyes, but these figures do not disprove this assertion, for allowance must be made for the predominance of hypermetropia over other forms of ametropia. Eyes that were highly hypermetropic were not found to possess a greater liability to the disease than the lower grade of this anomaly, for in no instance was the degree of hypermetropia higher than 3 D. The average degree was 1.66 D. *Myopia* occurred 28 times. The average excess in the length of the globe being 1.94 D. Twenty-one patients had *emmetropic* eyes. Twenty eyes were *astigmatic.* Of these the axis of the cylinders were vertically placed in 11 instances and horizontally in 7. Mixed astigmatism existed in 2 cases. From observations elsewhere the authors are confident that astigmatism against the rule occurs in the vast majority of cases of glaucoma, and it has been a great disappointment that this belief was not substantiated by the examination of this series of cases. As would be expected from the predominance of hypermetropia and the age at which most of the patients came under observation, glasses had been worn for varying lengths of time and repeated changings of the correction on account of failure to relieve symptoms were very frequently noted. The average length of time during which glasses had been worn was ascertained to be 11½ years for near, and 15½ years for far. In 86 cases where the refraction error and *the range of accommodation* were noted in conjunction, 30 showed an absolute loss of accommodative power out of proportion to the age of the subject. In the remaining 56 cases accommodative power was normal. Tension was increased to 63.15 per cent. of the cases in which there was normal accommodation whilst in 36 (88 per cent.) it was diminished.

From a consideration of these findings the authors have embodied the thoughts which have been engendered into *the following conclusions:*

Simple glaucoma occurs in either sex with about the same degree of frequency, but manifests a preference for the eyes of males. The majority of cases occur in the beginning of the fifth decade. It is quite uncommon, occurring in about .736 per cent. of the cases which seek treatment at ophthalmic hospitals. All forms of ametropia are equally liable to this affection. With the exception of articular rheumatism and influenza, which appear to induce changes that favor its development, there are no other particular systemic diseases which predispose to it. It is a binocular affection, although a period of 20 months usually intervenes between the manifestation of the symptoms in the two eyes. The two most prominent subjective symptoms are failing sight and headache, but neither of these possess characteristics which would serve to differentiate them from those occurring in other forms of occular disease. This form of glaucoma is slowly progressive, 2½ years being the average length of time required to induce blindness after the appearance of the initial symptoms. Signs of irritation in the anterior segment of the eye are usually absent, but 4.52 per cent. of the cases exhibit such changes. An inflammation of the optic nerve is a constant attendant upon glaucoma, being noted in every eye containing a pathologic excavation. It manifests itself as a low-grade neuritis affecting the entire structure of the nerve, and seems to render the nervous tissue more liable to the peculiar kind of excavation which is the most constant characteristic of glaucoma. No one of the four findings most commonly observed in glaucoma is essential to the disease, for the disease may occur without an excavation, without the field being contracted, without the diminution of central visual acuity, or without rise of tension. The excavation, however, is the most constant symptom, occurring in 81.43 per cent. of all the eyes which were examined. Although the excavation shows a marked predilection to occupy the temporal half of the disc, no part of the nerve escapes. In exceptional cases the excavation appears at the temporal edge of the disc as a continuation of the physiological excavation. From here it gradually spreads over the head of the nerve, encroaching upon its structure until only a narrow rim of nerve fibres remains at the nasal edge. Central visual acuity may remain normal although the field for form and color be encroached upon (in 10.77 of the cases.)

The tendency of the scleral ring to become visible all around the disc, and its disposition to broaden especially to the temporal side, are significant of the degree of intra-ocular tension to which the globe has been subjected, and go hand in hand with the extent and depth of the excavation. As the broadening of the ring however, usually appears before the excavation, its presence in eyes possessing other symptoms of glaucoma should always excite suspicion of this disease.

Opacities in the refracting media are found in an unusually large percentage (86.52 per cent.). These, however, are to be regarded as being more the result of senility than expression of the glaucomatous state. Arcus senilis (35.92 per cent.) and lenticular opacities (28.44 per cent.) are the most common, while corneal opacities, directly traceable to the increased intra-ocular tension, occurred in 20.38 per cent. of the eyes. Vitreous opacities occur in but few instances, and, originating in a choroiditis which complicates the disease, are not an essential feature of it.

The cornea is the most liable of the refracting media to be affected by the glaucomatous process. The increase in intra-ocular tension as determined by palpation is not necessarily a constant factor, being detected in 109 cases (32.63 per cent.). Where the tension is increased, the field will be distinctly cut or the nerve excavated. Rigidity of the sclera is often the first indication of increased, ocular tension. This rigidity may be accounted for either by a connective-tissue thickening in that tunic, or by an actual increase in the intra-ocular tension rendering the sclera more resistent to the examining finger. The shallowing of the anterior chamber exists in eyes where there is no excavation, but the converse is not true, for in every eye where there is an excavation the chamber will be shallowed. This would seem to show that the shallowing of the chamber occurred before the increased tension had excavated the nerve. As the chamber grows shallower and the tension higher the pupils will become larger, and the larger the pupil the less will there be reaction to light. This is not always true, however, for in 12 instances where the tension was distinctly elevated, and there were well marked excavations, the pupils were but 2½ *mm* in size and they responded perfectly to light and convergence stimuli. Other signs of increased intra-ocular tension, such as the choroidal halo and the venous and arterial pulses occurring in such a small proportion of cases show that they are not constant factors of the disease, and their absence cannot be regarded as negative evidence for the existence of a glaucomatous state. Increased tension limits the action of accommodation in only 34.88 per cent. of the cases. In the great majority of the cases the limitation of the field consists in a concentric contraction for color and form to an equal extent.

The consideration of the relative amount of contraction in the form and color field, often adopted in the distinction between an atrophic and a glaucomatous excavation, is valueless, as the findings show that in quite a large proportion (13.06 per cent.) of the cases the color field was relatively more affected than the form. Indeed in 16 of these 32 cases (50 per cent.) the form field was normal whilst that for red was contracted to 20 degrees or less. Contrary to the findings of other observers, the most frequent type of restriction of the visual field consists in concentric limitation of the entire field and not in the contraction to the nasal side. The limitations of the field in this latter position in 7.92 per cent. of the cases evidenced that this portion of it was peculiarly liable to be affected by the glaucomatous process, for the other portions were not equally affected by the disease. Full fields are not inconsistent with glaucoma, for in 129 eyes where the state of the tension, the degree of the visual acuity, the character of the excavation, and the extent of the field were noted, 30 eyes did not exhibit any lessening in the extent of their fields for form or for color.[1] "The effect of the administration of eserine and of the performance of iridectomy in checking the course of the disease is proportionately the same in the treatment of simple glaucoma. As operative procedures are always to be deprecated when other measures are equally valuable, eserine should be employed in all cases of the disease. If at the end of a month the extent of the fields has diminished, iridectomy should be resorted to, as there will be nothing further to expect from the action of the drug. If at the end of that time, however, an improvement is noted, as evidenced by a study of the field, the drug should be continued, as there is reason to expect that a beneficial action will be exerted for ten months upon the extent of the field and fifteen months upon the visual acuity. After iridectomy is performed there is ground to believe that the course of the disease will be checked for a period of 18 months in 50 per cent. of the cases. Eserine is powerless in 20 per cent., iridectomy in 10 per cent. of the cases. So that 10 per cent. of all cases of simple glaucoma will not be benefited despite all therapeutic measures which may be employed."

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[1] The observations which were made in regard to *treatment* have been published elsewhere (*Wills Eye Hospital Reports*, vol. i.), but for sake of completion the authors have incorporated the conclusions which they drew in that article.

AN UNUSUAL CASE OF HYALINE BODIES IN THE OPTIC NERVE.

By Dr. H. GIFFORD, Omaha, Neb.

(*With one drawing in the text.*)

THE ophthalmoscopic picture presented by a pronounced case of hyaline bodies in the papilla is so remarkable and is so puzzling to one who has never seen nor read of it, that it is unfortunate that so little consideration has been given it in the great majority of text-books. Until the recent publication of Panas's work, the only text-books in which I have found it mentioned are that of Noyes and the little one of Swanzy. Graefe-Saemisch and De Wecker-Landolt have but a few words on the subject, and the ophthalmoscopic works of Mauthner and of Loring have nothing. The best description of the ophthalmoscopic appearances which I have found is in De Wecker and Masselon's *Ophtalmoscopie Clinique* (2d ed., p. 214). They there describe a case as altogether exceptional, in which the whole papilla and a little of the adjoining retina were covered with these bodies, the vessels, however, not being covered at all. More marked than this case is one figured by Nieden[1] in which the greater part of the optic disc, with parts of the retinal vessels on it, was covered by a mass of hyaline bodies which bulged into the vitreous to a height of more than 4 *mm.* (14 D.). The case which I shall first describe far exceeds any hitherto reported, in an otherwise normal eye, in the extent of the affection, beside being unusual in other more important particulars.

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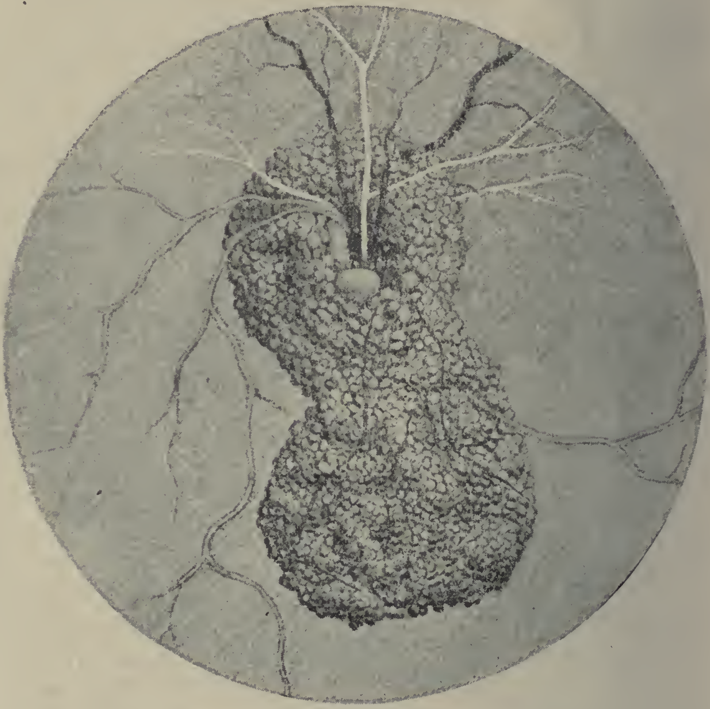
[1] Nieden, *Arch. f. Augenheilkunde*, xx., p. 73.

Sept. 16th, 1892, Zella K., a bright, healthy-looking girl eleven years old, came to my office with the following history: R. E. has been slightly turned in since infancy; nothing else unusual about eyes, until two years ago, when she had some trouble with the V. of both eyes and then noticed that V. of R. E. was much worse than that of L. E. At that time complained of slight pain in R. E. and it was examined by Dr. Thomas Kelly of South Omaha (through whose kindness the child was later sent to me), who found the right optic disc to be covered with hyaline bodies. Since then she has noticed no change in the eye except that V. has got worse.



Stat. praes. R. E. slight convergent strabismus, pupil reacts only on indirect stimulus; V. = doubtful perception of light; eye otherwise normal, except for slight vitreous opacities and the conditions to be described at and about the optic disc. These conditions can best be understood by reference to the accompanying figure, which represents the appearance of the upright image. The papilla and the surrounding retina, for a distance, in all directions, of about half the diameter of the average normal disc, was entirely concealed by a mass of hyaline bodies, the summit of which was 9 D above the rest of the fundus; below the papilla, this mass, after a slight constriction, was continued into another of about twice the diameter of the disc. The hyaline bodies were translucent, of somewhat pearly lustre, reminding one of half-soaked-up grains of tapioca. Near the centre of the disc, they seemed to have coalesced into two unusually large bodies. Near the temporal side of the main mass, some isolated whitish spots, probably, but not positively, of the same nature, could be seen either in the outer layers of the retina or the inner layers of the choroid. From the crater-like depression in the centre of the main mass, the superior and supero-temporal vessels could be seen emerging, while the remaining vessels ran through the mass for some distance before coming into view. The most remarkable thing about the vessels was the fact that the superior, supero-temporal, and supero-nasal arteries were entirely occluded and changed into glistening white bands from their points of emergence to a considerable distance out into the retina.[1] The supero-nasal branches were white to their terminations, while the superior and supero-temporal branches regained some color, though not a normal appearance, at from 4 to 5 papilla-breadths from the centre of the disc. In the vitreous, mostly in the posterior part, were a number of fine slightly reddish opacities evidently from comparatively recent hemorrhages. The rest of the fundus showed nothing abnormal. The other eye was normal throughout, unless a slight pallor of the outer half of the disc was pathological; V $\frac{20}{30}$ + H = 1.5 D; field and color-sense normal. Dr. Kelly, who is a competent ophthalmoscopist, assures me that when he first saw the case the vitreous was perfectly clear and that he noticed none of the changes in the arteries which were so marked at the time of my first examination. In the two years during which the patient has been under my observation, the vitreous opacities have cleared up entirely or nearly so, the mass of hyaline bodies has grown somewhat larger; and white lines have begun to appear along one of the arteries not, I think, affected when I first saw the eye. During the last eight months there has grown from the lower of the larger masses, at the edge of the central depression, a body, apparently of the same substance as the other hyaline bodies, but differing in shape from anything which I have seen described in this connection. When first seen six months after the next preceding examination it was somewhat trumpet-shaped, the flaring end, nearly a half papilla-breadth in diameter, projecting out into the vitreous and containing a deep depression, while the tapering end was attached to the large hyaline body at its central edge. Within the last two months the shape of this growth has changed so that it is now irregularly cylindrical, the end which projects into the vitreous being bluntly conical, like a revolver bullet, and apparently hollow. I have not attempted to have the sketch altered to represent this growth.

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[1] At a recent examination I have found that these white vessels are not entirely obliterated, but have a very small blood stream in the centre of each one. It is quite possible that I overlooked this in earlier examinations.

This case is unique in several particulars. The only reported case which compares with it in the extent of the affection is that of Remak.[1] He observed, in a patient with retinitis pigmentosa, a series of clumps of hyaline bodies which had obliterated the papilla and extended for some distance into the retina. This case, however, could not be examined with perfect accuracy, as the eye had nystagmus.

The changes observed in the vessels, in the case of my patient, are even more unusual than the extent of the affection. Where the hyaline bodies exist as a complication of retinitis pigmentosa, it is, of course, common to find the vessels much reduced or even, as in Remak's case, nearly absent from the entire retina; but aside from this, in the other cases hitherto reported, the vessels, though sometimes partly covered, have shown no changes that could in any way be connected with the hyaline bodies.

Another unusual feature in the case is the age of the patient. So far as I can learn the youngest patient hitherto described with hyaline bodies in the optic nerve is one of Ancke's[2] who was fourteen years old, the eyes also being affected with retinitis pigmentosa. Excluding eyes with retinitis pigmentosa, the youngest patient that I find reported is one of Stood's,[1] a seventeen-year-old girl. My patient was first found to have an enormous development of these bodies at the age of nine years.

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[1] Remak, *Centralblatt f. Augenheilkunde*, ix., p. 257.

[2] Ancke, *Centralblatt f. Augenheilkunde*, ix., p. 167.

Whether in this case the blindness was due solely to the development of the hyaline bodies, or whether the development of the latter was simply favored by some foregoing disease of the optic nerve, is impossible to decide.[2] Be this as it may, the changes which have taken place in the vessels and which appear still to be going on, to some extent, indicate that we ought to take with some reserve the statement of Terson,[3] that the prognosis, so far as the effects of these bodies themselves are concerned, is absolutely good. One of Nieden's patients had a reduction of vision, probably due to the effects of the hyaline bodies alone ; and another case, which I have observed, also gives the same indication, besides having other points of interest. This was the case of J. D., a robust-looking man about forty-five. For a year or two he had noticed a blur coming suddenly before the eyes, when looking intently at anything. I found the vision to be R. E. = $\frac{20}{50}$ — L. E. = $\frac{20}{40}$ —; no improvement by glasses; fields normal for white but R. E. shows decided contraction for blue, yellow, and red; and L. E. for blue and yellow. Vision blurs unusually quickly during these tests and statements become conflicting. The only condition to account for the defects of sight, was a marked development of hyaline bodies in both optic discs. In the right eye, these bodies covered the whole nerve and all its vessels with the exception of a small area in the centre which was free from them. In the left eye, the conditions were much the same, except that the free area in the centre of the disc was larger. In all other respects, the eyes were perfectly normal. This man was apparently in robust health and as there was nothing in the case to indicate any kidney trouble, I am not sure that the urine was examined. If it was, no abnormality was found and no record was made of the test. About a year later, however, he developed symptoms of Bright's disease and died at about the end of another year.

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[1] Stood, *Zehender's Monatsblätter*, 1883, p. 512.

[2] Stood's two cases are supposed to show a relation between optic neuritis and hyaline bodies in the papilla ; but as in one case the bodies were found less than four weeks after the visual disturbance began, they had probably existed long before the neuritis occurred ; while in the other case, it is not at all certain that there had ever been a neuritis.

[3] Terson, *Archives d'Ophtal.*, June, 1892, p. 374.

If the eyes of this man had been examined, microscopically, by some one who had not seen him before his kidney trouble developed, the case would have been reported as one of hyaline bodies in connection with Bright's disease; whereas, in all probability, these bodies had existed in the nerves for some time before the kidneys were diseased. Hitherto, Bright's has been supposed to stand in some causal relation to these bodies on the strength of the case of Gurwitsch[1]; but it seems to me that this case proves even less with regard to any such connection than the case just described. In examining the eyes of a patient who had died of nephritis, he found hyaline bodies in one nerve-head (not in the other), but there was no evidence to show whether the nephritis or the hyaline bodies had had the earlier origin.

With regard to the controversy as to whether these bodies, as occurring in the nerve-head, are related to the similar bodies which grow from the elastic layer of the choroid, it is evident from microscopic examinations that they often occur at some distance from the main ending of the lamina elastica, at the periphery of the nerve. But if, as Kuhnt asserts, this layer sends off fine prolongations into the substance of the nerve, it is quite possible that the bodies take their origin from these prolongations. Hirschberg and Cirincione,[2] who have been the main upholders of the view that these bodies, in the nerve-head, are of an entirely different nature from the "Drusen" of the choroid, insist upon the fact that the latter are covered with epithelium, and sometimes contain epithelial cells in their centres, while the "Drusen" of the nerve-head show nothing of the kind. This difference, it seems to me, can be explained by the relation of the "Drusen" of the choroid to the epithelial layer of the retina. The latter, lying as it does in immediate contact with the lamina elastica, would naturally form a covering for the excrescences from the latter, and might easily have some of its cells incorporated into them by being pinched off between two or more " Drusen " in the process of coalescing. In the nerve-head, of course, there is no chance for any thing of the kind to occur. On the other hand, it seems that the one pathological process which stands in an undoubted relation to the causation of these bodies, in the nerve, is retinitis pigmentosa, and in this disease, also, " Drusen " in the choroid are extremely common.

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[1] Gurwitsch, *Centralblatt f. Augenheilkunde*, August, 1891.

[2] Hirschberg and Cirincione, *Centralblatt f. Augenheilkunde*, June–July, 1891.

But whatever the exact starting-point of these bodies may be, it is evident that they do not grow simply by the degeneration of the pre-existing tissues of the part ; nor does it seem likely that they are of the nature of excretions, for there are no structures in the nerve-head to excrete them. Apparently, given, from whatever cause, a nucleus for one of these bodies, it grows, within certain limits, by deposition on its surface from the fluids of the body ; something as a crystal increases in size, when hung in a solution of the same substance.

Finally a word as to a matter of priority. Hirschberg mentions the case described by himself and Cirincione as the first in which test of vision, ophthalmoscopic examination, and microscopic examination had been made ; but it is evident from a comparison of his text with that of De Schweinitz[1] that it is to the latter's case that this distinction properly belongs ; for in Hirschberg's case, although the ophthalmoscope was used, it is plain that the hyaline bodies could not be seen, on account of a turbid vitreous.

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[1] De Schweinitz, *Trans. American. Oph. Soc.*, 1892.

AN ORBICULARIS PUPILLARY REACTION.

By Dr. H. GIFFORD, Omaha, Nebraska.

FOR some years I have been observing a pupillary reaction of which I have found no mention in the ophthalmic and physiological literature at my disposal. The only reactions noted in the larger handbooks, as well as in the most recent text-books, such as those of Panas and of Roosa, are, first, contraction on exposure to light and on efforts of accommodation and convergence; second, dilatation on the withdrawal of light and on the stimulation of the sensory nerves in various parts of the body, as well as in certain emotions.[1]

The reaction to which I refer is a contraction of the pupil which takes place when a forcible effort to close the lids is made. This, for reasons to be given later, can be best observed on a subject blind, or nearly so, from some disease of the retina, optic nerve, or brain. When such a subject, with the eyelids held apart by the observer, is told to shut the eyes tightly, the pupils, if the eyes are not rolled up too sharply, are almost always seen to contract perceptibly, and sometimes very markedly. Sometimes the eyes are rolled up so far that the pupil is not visible while the contraction of the orbicularis is going on, but as soon as the latter is relaxed and the pupils come down into view they are seen, for an instant, to be contracted, though they dilate again promptly to the average, or rather more than average size. With subjects having good vision, this reaction often cannot be observed, except under special conditions, because the contraction maintained by the light of an ordinary well-lighted room is greater than that caused in most eyes by the orbicularis contraction; therefore it is best to use the dark room, having enough light come from one side to allow the changes in the pupil to be easily observed. Under these conditions the reaction mentioned can be seen plainly in the great majority of eyes; in a few it is very slight, while in a still smaller percentage it is so slight that it is questionable whether it occurs at all. As before mentioned, this contraction of the pupil is best demonstrated by having the subject try to close the lids tightly while they are forcibly held open (it is better to hold the lids of *both* eyes open during the test); but in many people this holding open is not necessary; if they simply shut the lids tightly, the pupils, when the lids are opened quickly, are seen to be contracted, though not so much so as when the lids are held open; this, however, requires a quicker and more careful observation than when the lids are held open, for the sudden access of light often causes a still sharper contraction of the pupil than that caused by the orbicularis reflex.

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[1] The cortical reflex described by Haab (*Jahresbericht f. Ophthalmologie*, xxii., p. 51) is not mentioned in any ordinary text-book that I have seen, but is thoroughly considered in Knies's work, *The Eye in General Diseases.*

I speak of this pupillary contraction as the orbicularis reaction, mainly for the sake of brevity, since it will be difficult or impossible to prove that the stimulus for the pupillary contraction comes solely from the orbicularis centre. It is probable, I think, that it is simply the result of an overflow stimulus; that is, the intense activity which the attempt to close the lids excites in the cells of the nucleus of the orbicularis fibres of the facial, overflows, so to speak, into the cells of the pupil-contraction-centre. That this reaction is not merely the result of a stimulation of the convergence centre is shown by the fact that, in many cases, when the attempt to close the lids is made, the eyes can be plainly seen to be slightly divergent. Generally, when the subject is told to shut the eyes tightly, the eyes turn up and make a conjugate lateral movement to one side or the other, or alternately from one side to the other; more rarely there is some convergence at first, but in nearly all cases, if the attempt to close the lids is continued, the axes become more or less divergent (the pupil-contraction continuing meanwhile), and remain so until the orbicularis contraction is relaxed. The fact that, in some of the cases in which the orbicularis pupil-reaction could be observed but slightly, or not at all, the patients, when told to shut the eyes, kept looking straight ahead instead of rolling the eyes up, made me suspect that the pupil-contraction might depend upon the action of the superior rectus; and I have found, as a matter of fact, that, with some people, a slight pupil-contraction does take place when they simply look up sharply; but this superior rectus pupil-contraction does not begin to equal in constancy or extent that occurring with the orbicularis contraction.

It is more difficult and perhaps, for most cases, impossible to prove that the orbicularis contraction does not cause the pupil to contract solely by exciting the action of the accommodative muscle, the latter then exciting the pupil-sphincter. But, in some cases, it can be clearly shown that this is not the case, since with these patients the utmost exercise of the accommodation and convergence fails to produce as marked a contraction of the pupil as that caused by the forced attempt to close the lids.

For example, in the case of Z. K., a girl of thirteen, blind in the right eye, but with vision $\frac{20}{20}$—and normal accommodative power in the left eye, the pupils contract only to about 6 *mm* in diameter when objects are fixed at the near point or nearer, while the forced action of the orbicularis causes them to contract to about 3 *mm*. I have seen several similar cases, some of them in patients with both eyes normal. In the great majority of cases, if the test is made by good daylight, or even by gaslight coming from the side in a dark room, the pupil-contraction accompanying convergence and accommodation is greater than that caused by the orbicularis contraction; but if the light is made as dim as is consistent with accurate observation, it will every now and then be found that the near-vision contraction does not equal the orbicularis contraction. This indicates that, while the orbicularis reaction is independent of the light stimulus, the contraction of the pupil accompanying near vision does not reach its maximum except by good light.[1] An additional proof of the independence existing between the contraction of the pupil caused by the orbicularis, and that accompanying the contraction of the ciliary muscle, I have found in several pathological cases, where no perceptible contraction of the pupils occurred on stimulus from light, convergence, or accommodation, while the orbicularis test produced a distinct though slight contraction of the pupil.

I have referred to the fact that when the person observed looks straight ahead, while trying to close the eye, the pupil-reaction is less marked, or not at all observable. At least a partial explanation of this is to be found in the independent action of the different parts of the orbicularis muscle; the parts which control the lids proper can act with entire independence of those which pull down the eyebrows and pull up the tissues of the cheeks. Now, the orbicularis pupillary reaction seems to be mainly, if not wholly, dependent on the action of the parts which move the lids alone, and some people, while apparently trying hard to shut the eyes when the lids are held open, really make little or no effort with the strictly palpebral portions of the orbicularis, but expend all their energy on the more peripheral portions of the muscle, and this is especially true if the gaze is kept fixed on some object straight ahead. I judge that it is chiefly the palpebral portions of the muscle that cause the pupil reaction, because when experimenting with myself, if, with the lids held apart, I strongly contract the more peripheral portions alone, I find that I can easily keep my eyes directed at any point which I choose to look at; and under these circumstances (as my colleague, Dr. Overholt, tells me) there is not only no contraction of the pupil, but even, at times, a slight dilatation; while, if I strongly contract the palpebral portions alone (the lids being held open), I find it impossible to keep looking straight ahead, the eye turns up, and the pupil contracts sharply. I have also found this connection between the pupil-reaction and the strictly palpebral portions of the orbicularis to obtain in several other cases which I have examined with especial reference to this point, but it is not every patient that has the intelligence to make the test satisfactory.

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[1] This may be due, in part, to the imperfect vision by the dim light. Wolffberg has recently asserted that the extent of the pupillary contraction, in near vision, varies directly with the acuity of vision obtained by the accommodative effort which causes the contraction.

With regard to the importance of this pupillary reaction from a pathological standpoint, I can say but little. It sometimes persists when all other non-medicinal myotic reactions fail, but as it occasionally cannot be demonstrated in normal subjects, it will be difficult to give to its absence a distinct pathological significance. The reaction is of use in determining whether the pupil-sphincter is paralyzed, in cases where the ordinary tests fail. In several cases of suspected brain tumor, for instance, where the pupils showed no contraction on light, convergence, or accommodation (the failure of the last two being probably, more or less, due to the poor sight), I have found the pupils to contract promptly on forced contraction of the orbicularis. In similar cases, it may also serve to eliminate the possibility that the dilatation of the pupil is due to a mydriatic having been used by some previous observer.

From a physiological standpoint this pupil-reaction is interesting as tending to confirm either the views of Mendel (*Berlin. klin. Wochenschrift*, 1887, 48, p. 913), who claims that in rabbits the fibres of the facial which supply the orbicularis, arise, not from the common facial nucleus, but from the posterior part of the oculomotor nucleus, or those of Bechterew (cited by Noyes, *Diseases of the Eye*), who asserts that special fibres connect the nucleus of the oculomotor with that of the facial nerve. The fact that the reaction seems to depend more particularly, if not entirely, on the action of the palpebral portions of the orbicularis, indicates, perhaps, that, in man, it is only the nerve fibres supplying these portions, which have an oculomotor origin; or, at least, that the nuclear cells of these fibres are most intimately connected with the main body of the oculomotor nucleus.

TREATMENT OF SYPHILITIC AFFECTIONS OF THE EYE BY MERCURY, POTASSIUM IODIDE, AND PILOCARPINE COMBINED.[1]

By G. HERBERT BURNHAM, M.D., F.R.C.S. Edin., M.R.C.S. Eng.,

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July, 1894, a man consulted me suffering from syphilitic inflammation of both eyes of nine months' duration at least. His physician had been treating him for acquired syphilis since December, 1893. Three months or so prior to this his eyes became affected while under the supervision of another physician. From December, 1893, to July, 1894 he had received antisyphilitic treatment in the form of mercury and the iodide of potash. Atropine was also used locally during the entire winter. This latter remedy had not been used for two weeks prior to consulting me.

The condition of the eyes July, 1894, was as follows:

Left eye.—Much conjunctival, subconjunctival, and ciliary injection, pain at times, aqueous turbid, many lymph dots on the posterior surface of the cornea, very many posterior synechiæ, some being broad and dense, with the thin deposit of lymph in the pupillary area. V = perception of light only.

Right eye.—No letters of Snellen type at twenty feet, and only letters of No. XL. of the same type at eight inches; cornea and aqueous very slightly affected, posterior synechiæ more numerous and broader so as almost to be without a break, *i. e.*, so as to form almost one solid ring of adhesion, and the lymph deposit in the pupillary area denser. He said that his vision was now much worse than three months previously. I continued the administration of mercury and the iodide of potash internally, and atropine locally. At the end of four weeks no improvement had taken place, in fact the eyes were slowly getting worse. I now made a change in the treatment as follows : I still continued the internal use of mercury and the iodide of potash, but added pilocarpine, giving it hypodermically. I began with gr. $\frac{1}{8}$, and gradually increased it to gr. $\frac{1}{4}$. I continued this for three weeks, and then again in three weeks for two weeks more, and again in five weeks for two weeks longer. While in use the pilocarpine was given every day, unless its administration gave rise to nausea, headaches, or oppression over the region of the heart, when it was stopped for one or two days, or perhaps the dose reduced only. After one week an improvement could be seen ; at the end of three weeks it was quite evident, and at the termination of fifteen weeks or so the vision was $\frac{20}{50}$ Snellen.

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[1] Read before the Toronto Medical Society, February 7, 1895.

The posterior synechiæ had been steadily thinning so that the broad dense bands began to look quite attenuated and weblike.

An interval of rest of eight weeks was given, the mercury and the iodide of potash, however, being continued. I may here mention that he has steadily taken the mercury and the iodide since he came under my care. At the expiration of this time the pilocarpine was again given hypodermically for ten injections. Some of the posterior synechiæ above mentioned have now given way, and such an alteration has shown itself in those that remain, that I feel assured that by persistence in treatment all these iritic adhesions will be removed. The deposit of lymph, once markedly present in the pupillary area, seems to be practically gone in the left eye and much lessened in the right. The vision is $R = \frac{20}{40}$, $L = \frac{20}{20}$.

I may also mention that since the pilocarpine was given, the improvement, that then began, has been uninterrupted.

The progress of this patient under the combined treatment has been most interesting to me. It has not only shown the apparent inertness of the powerful remedies, mercury and the iodide of potash, but also the immediate and brilliant results of the combined use of mercury and the iodide of potash internally and pilocarpine hypodermically.

We all know that the vigorous and long-continued use of mercury and the iodides has too often had far from a satisfactory effect not only in syphilitic affections of the eye, but also in syphilitic lesions in other parts of the body, as I think has again been exemplified in this case. The addition of pilocarpine, however, produced such a markedly beneficial effect that I feel justified in claiming for this combined form of treatment a position as a most valuable and important addition to our means of treating syphilitic affections of the eye, and if of the eye, why not of any other part of the body that may be affected. From the manner in which this combined form of treatment has acted upon long-standing iritic adhesions, I feel it ought to be exceptionally useful in syphilitic lesions of long duration.

Pilocarpine has been used to a limited extent in syphilis; but I have never heard of its employment combined with mercury and the iodide of potash as is now brought before the attention of the profession.

It is not from this case alone that I have drawn my conclusions; for others, not so typical, have, however, all contributed to strengthen my belief in its efficacy.

The above mentioned treatment has ever since been carefully followed out, and is still being continued with such gratifying results, that with permission, I purpose later on publishing them in this journal.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FOURTH QUARTER OF THE YEAR 1894.

By Dr. ST. BERNHEIMER, in Vienna ; Prof. C. HORSTMANN, in Berlin ; and Dr. P. SILEX, in Berlin ;

WITH THE ASSISTANCE OF

Dr. S. M. Burnett, Washington ; Dr. Dantone, Rome ; Dr. Herrnheiser, Prague ; Prof. Hirschmann, Charkow ; Dr. P. von Mittelstädt, Metz ; Dr. Sulzer, Geneva ; Dr. Werner, Dublin ; Dr. C. H. A. Westhoff, Amsterdam ; Dr. Schiötz and Dr. Ole Bull, Christiania ; Dr. R. Greeff, Berlin ; Dr. Deus, Berlin ; and others.

Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS. STATISTICAL REPORTS.

1. Puccioni. The eye clinic of the Royal University of Rome in the year 1893–94. *Boll. d'Ocul.*, xvi., 13, 15, p. 101.

2. Vienna. *Year-Book of the Imperial Hospitals for 1893.* Eye departments, p. 276 *et seq.*

3. Bjerrum. Mechanism of accommodation. *Med. Aarsskrift.*, Copenhagen, 1894.

4. Greeff. The retina of the vertebrates. Investigations with the Golgi-Cajal silver-precipitation method and Ehrlich's

methylene-blue stain. A translation and collection of the works of Ramon y Cajal, done with the assistance of the author. Bergmann, Wiesbaden, 1894.

5. HAAB. Atlas and elements of ophthalmoscopy and ophthalmoscopic diagnosis. *Lehmann's Medical Hand Atlases*, No. vii., 1894.

In the Rome (1) clinic, according to PUCCIONI, 95 patients were admitted, 34 extractions being done, and 778 patients treated as out-patients. DANTONE.

In the public hospitals of Vienna (2) in 1893 855 cataract extractions were done ; 800 with good success, 39 with moderate, and 15 with bad result. One patient died. 263 iridectomies were made for optical purposes, 93 for glaucoma, and 13 as preparatory operations, and 123 eyes were enucleated. HERRNHEISER.

GREEFF (4) has undertaken the laborious work of translating, arranging, and annotating the rather inaccessible and extremely valuable papers of Ramon y Cajal on the retina. The introduction by the translator, treating of the Golgi-Cajal and Ehrlich methods, is very welcome. The excellent original illustrations increase the value of the book, which has been well gotten up by Bergmann. The different chapters treat of the retina of the teleosts, frogs, reptiles, birds, and mammals. It is an invaluable book for him who would keep posted on the present status of our knowledge of the retina.

HAAB'S atlas (5) is an excellent work and is cheap. The selection of the illustrations is good, and the short descriptions are adequate. Although in artistic execution the plates are inferior to Jaeger's, its fulness and cheapness will recommend it both to the student and the practitioner.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

6. CUÉNOUD. Clinical bacteriology and parasitology of the lids. *Thèse de Paris*, 1894.

7. BLOCK. The healing of corneal wounds under the use of cocaine. *Medisch. Weekblad.*, 1894.

The various staphylococci are frequently found on the lid margins, particularly at the insertion of the cilia. CUÉNOUD (6), however, has never found streptococci here in the healthy lid. The most frequent lid affections are caused by staphylococci.

The Staphylococcus aureus is found in ulcerous blepharitis and hordeolum, the Staphylococcus albus in dry blepharitis. SULZER.

BLOCK (7) made experiments on the influence of cocaine on the healing of corneal wounds. In opposition to Mellinger, he found that cocaine had no injurious influence.

III.—INSTRUMENTS AND REMEDIES.

8. GUAITA. Formol in ophthalmology. *Ann. di Ottalm.*, xxiii., 5, p. 360 ; and *Lo Sperimentale*, xviii., p. 33.

9. DANESI. An antiseptic medicament in ocular surgery. *Boll. d'Ocul.*, xvi., 13, p. 100.

10. OSTENGO. Sea-bathing for ophthalmias. *Ibid.*, xvi., 14, p. 108.

11. VACHER. On permanganate of potassium in ocular affections. *Ann. d'ocul.*, cxii., p. 419.

12. DARIER. Subconjunctival injections once more ; their theory, practice, indications, and contra-indications. *Ann. d'ocul.*, cxii., p. 381.

13. ARMAIGNAC. On the employment of antiseptics, caustics, and anilin dyes in ophthalmology. *Soc. de méd. et de chirurg. de Bordeaux*, Oct. 12, 1894. *Ann. d'ocul.*, cxii., p. 340.

14. BRUN. On the disinfection of the conjunctival sacs. *Presse méd.*, Oct. 27, 1894.

15. ANDOGSKY. On the employment of formaldehyd for preserving eyes for operative work. *Wratsch.*, No. 41, 1894.

16. PATOJENKO. The action of scopolamine hydrobromate on the healthy and diseased eye. *Teschenedjelmk*, No. 27, 1894.

17. DUJARDIN. A new fixation forceps for eye operations. *Ann. d'ocul.*, cxii., p. 396.

18. SCHÖBL. Optotypes—for Bohemian physicians. *Franz Deutike*, 1895.

19. GROVE. Graduation of the arc on Javal's ophthalmometer and the graduation on a straight arm. *Annals of Ophth. and Otol.*, Oct., 1894.

20. KUNN, CARL. Suggestion for a modification of the ophthalmoscope. *Wiener klin. Rundschau*, No. 2, p. 19, 1894.

21. TROMPETTER. A lid elevator for cases of entropium. *Zehender's klin. Monatsbl.*, xxxiii., p. 30.

22. JACKSON. Value of the ophthalmometer in practical refraction work. *Trans. Amer. Ophth. Soc.*, 1894.

23. BAKER, C. H. Results of subconjunctival injections in some eye diseases. *Annals of Ophth. and Otol.*, Oct., 1894.

24. PRENTICE. On the practical execution of ophthalmic prescriptions involving prisms. *Amer. Journ. of Ophth.*, Jan., 1895.

GUAITA (8) considers formol an important antiseptic. Strong solutions kill the microbes, and weaker solutions prevent the development of cultures. Irrigation with a 0.2 % solution produces asepsis of the conjunctiva often lasting twenty-four hours, which is not obtained by the use of other antiseptics—even bichloride. DANTONE.

VACHER (11) uses 0.1 %-0.2 % solutions of permanganate of potash for irrigating the conjunctiva, nose, and auditory canal when these are the seat of excessive suppuration. Such solutions are also useful when used as injections through the tear passages. SULZER.

Without giving new case histories, DARIER (12) presents a condensed statement of the present status of the question of subconjunctival injections of bichloride. The different theories of absorption, the objections to the method, and its indications and contra-indications are all fully treated.

ANDOGSKY (15) preserves enucleated eyes by keeping them for five to seven days in a 0.5 %-0.1 % solution of formaldehyd, when they may be used for operation courses; the transparency of the cornea and the tension of the eye being preserved. HIRSCHMANN.

Scopolamine, according to PATOJENKO (16), is more energetic, but more transitory in its action than atropin. Its power of easing pain is considerable. It causes no symptoms of irritation. Eserin contracts the pupil more readily after scopolamine than after atropin. A solution of 1:1000 is sufficient. HIRSCHMANN.

In the paper GROVE (19) discusses the construction of the ophthalmometer: (1) the finer markings on the arc indicating the radius of corneal curvature as constituting an arithmetical series; (2) the graduation of the arc for double movable mires; (3) the graduation on a straight arm for a single movable mire; (4) the graduation on a straight arm for double movable mires. BURNETT.

JACKSON (22) finds the ophthalmometer useful as an approximative test. In only 6 % of the 500 eyes examined by him did the total astigmatism correspond with the corneal in both degree and direction of its axis. In 16 % additional the corneal and total astigmatism was the same in degree, only the difference was 0.25 D in 21.6 %; 0.5 in 20 %; and so on up to 1.5 to 3.5.

BURNETT.

BAKER (23) has used subconjunctival injections of bichloride with success in cases of interstitial keratitis, choroiditis, and chorio-retinitis. He uses a very weak solution, 1 to 30,000.

BURNETT.

PRENTICE (24) pointed out some time ago the law that the decentration of a lens for 1 *cm* gives a prismatic action of 1 prism diopter (△) for every diopter of lenticular refraction. This paper is a further amplification of that law, with the suggestion that, for the weaker lenses, from 0.25 to 2 D, the diameter of the lens be 6 *cm* in order to increase the possibility of use of such lenses in decentration for prismatic effect. BURNETT.

IV.—ANATOMY.

25. LUGARO. On the origin of the fifth, sixth, seventh, and eighth cranial nerves. *Arch. di Ottalm.*, ii., 3–4, p. 116.

26. BELLI. On the distribution of neuroglia in the optic-nerve chiasm in man. *Ibid.*, ii., 1–2, p. 19.

27. RETZIUS. Investigations on the ciliary ganglion. *Hygiea*, No. 9. *Svench. Läkarevell. Förhandl.*, p. 154, Stockholm, 1894.

28. NICOLAI. Form of the anterior surface of the lens. *Medisch. Weekblad*, 1894.

BELLI (26) describes the relations and the appearance of the processes of the neuroglia cells in the arched zones of the human chiasm. DANTONE.

RETZIUS (27) has found that the ciliary ganglion is a true sympathetic ganglion like the other ganglia of the head.

SCHIÖTZ.

NICOLAI (28) has endeavored to get the true form of the anterior surface of the lens. For this purpose he constructed an instrument by means of which he could measure a deflection of $\frac{1}{10}$ *mm*, and he also made plaster casts of pigs' lenses. He found that the relaxed lens is regularly curved. WESTHOFF.

V.—PHSIOLOGY.

29. SPALITTA. Effects of extirpation of the ganglion of Gasser, after the destruction of the superior cervical ganglion. *Arch. di Ottalm.*, ii., 1–4, p. 37.

30. FERRI. On the size of the field of view in the ophthalmoscopic examination. *Ann. di Ottalm.*, xxiii., 3–4, p. 180.

31. FERRI. Determination of the angle α with the perimeter. *Ibid.*, p. 175.

32. ANGELUCCI. The visual function of the aged, and its effects on the employment of color in painting. *Arch. di Ottalm.*, ii., 1–5, p. 3.

33. GULLSTRAND. A photographic ophthalmometric method and the results obtained with it, together with a method of clinical determination of the curvature of the cornea in its periphery. *Hygiea*, No. 8. *Svënsk. Lakeresellsk. Förhandl.*, p. 128, Stockholm, 1894.

34. REBOUD. The position of rest of the eyes. An experimental study. *Arch. d'opht.*, xiv., 11, p. 681.

35. OSTWALT. Ophthalmotonometric studies. *Graefe's Archiv*, xl., 5, p. 22.

36. GRAEFE, ALFRED. Accommodation and convergence. *Ibid.*, xl., 5, p. 247.

SPALITTA (29) reports various experiments made to determine the influence of the trigeminus on the nutrition of the coats of the eye, after destruction of the cervical sympathetic. DANTONE.

FERRI (30) again describes his method of measuring the extent of the fundus seen in the direct and indirect ophthalmoscopic examination. This method differs from that of Helmholtz, and the resulting values are greater. DANTONE.

ANGELUCCI (32) reports on the examination of the eyes of 100 individuals between the eyes of 60 and 80, as well as 14 painters, in respect to acuteness of vision, light sense, and color perception, and discusses in an intelligent manner the influence of the age of a painter on the chiaroscuro and coloring of his pictures, which often differ from those painted in early life. Many painters are mentioned whose late work showed much improvement on their earlier work, and his statements in regard to them are very interesting. DANTONE.

GULLSTRAND (33) gets his illumination with two very strong lamps, and photographs the corneal image of an illuminated black disc with white concentric circles arranged in such a way that the size of the corneal image is directly proportional to the radius of the surface. Thus a single photograph gave 7 measures of the radius of curvature of the cornea in every meridian, and it was found that the applanation is relatively slight in the centre of the cornea, while it is more marked in the marginal zones, and is asymmetrical in both the vertical and horizontal meridians. The sum of the applanation up and down is greater than the sum of the applanation outward and inward. SCHIÖTZ.

Following Snellen's method, REBOUD (34) determined the position of rest, taking also into account refraction and acuteness of vision, in a number of soldiers (P = parallelism ; C, convergence; D, divergence). In E., P and D predominate ; with diminished acuteness of vision in one eye, a tendency to C ; with further diminution, a tendency to D. In H., with moderate or diminished acuteness of vision, there is mostly C, due to accommodative effort. In M., with good sight, there is also C, due, according to the author, to the frequent existence of astigmatism, leading to accommodation in the effort to increase the acuteness of vision. In H. A., even with good V, the tendency is mostly to P or D. In M. A., C is more frequent than in H. A., but there is no fixed rule. In anisometropia, C is frequent. Therefore, P and D are the positions of eyes which see without a strain, or in which V is poor ; while particularly in the accommodating eyes of H., C is present. The length of the basal line had no influence on the position. In all refractive conditions, vertical deviations were found, and the position of one eye frequently differed from that of the other, particularly in cases of anisometropia, while good and equal sight in the two eyes favors a similar position. Diminution of V, or the strain to increase V, leads readily to the abandonment of binocular vision and then to squinting, in the development of which latter the determination of the position of rest is of little importance. V. MITTELSTÄDT.

OSTWALT'S (35) ophthalmotonometric investigations led him to the conviction that measurements made with Maklakoff's tonometer have a more objective character than those made with Fick's instrument. For absolute measurement Fick's tonometer is preferable, provided that the observer is somewhat practised in its use. The author believes, moreover, that the inadequate method of Bowman, of determining tension by the touch, should give place to the use of a tonometer.

Sections VI.–XI. Reviewed by Prof. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

37. Ricchi. The anomalies of vision in 45,000 observations. *Boll. d'Ocul.*, xvi., 9. p, 55.

38. Niebuhr. A contribution to the operative treatment of myopia. *Inaug. Dissert.*, Halle, 1894.

39. Chauvel. Ophthalmoscopic studies. Hyperopia. *Rec. d' opht.*, 1894, p. 573.

40. Raia. A summary exposition of the causes, effects, and treatment of asthenopia. *Ann. di Ottalm.*, xxiii., 3-4, p. 222.

41. Miles, H. S. The errors of refraction found in four thousand eyes. *The Refractionist*, Oct., 1894.

42. Howe. Note on lid pressure as a cause of astigmatism. *Amer. Journ. of Ophth.*, Sept., 1894.

43. Theobald. Some typical cases of subnormal accommodative power. *Trans. Amer. Ophth. Soc.*, 1894.

44. White, J. A. The practical value of low-grade cylinders in some cases of asthenopia. *Ibid.*, 1894.

Niebuhr (38) reports eighteen operations for myopia done in the clinic at Halle. Discission was first done, then the opaque lens extracted by the linear method, and finally the posterior capsule divided. In only three cases was the swelling of the lens accompanied by irritation or pressure symptoms ; with the extraction there was prolapse of iris three times, loss of vitreous twice, and hemorrhage into the anterior chamber once. In one case eczema of the lid margin and injection of the conjunctiva developed under the bandage, followed by iritis and hypopyon. The eye was lost in another case from infection of the cornea due to a croupous conjunctivitis. The difference in refraction was 18 D on an average, all the eyes being finally hyperopic. The acuteness of vision was increased in all but three cases.

Of 720 hyperopes with poor vision that Chauvel (39) examined 53. 4% had poor sight in both eyes ; 21. 2 % in the right only ; and 15. 4 % in the left only. No more than half the number had an equal amount of hyperopia in the two eyes. All the individuals examined had vision below the French military standard, which is ½ for one eye and $\frac{1}{10}$ for the other. Careful investigation was made as to the cause of the poor vision. Examinations with the stenopaic slit showed that the refraction *per se* played only a minor rôle. Frequently there were anomalies of the field of vision, mostly a more or less marked concentric contraction usually proportional to the amblyopia. Notwithstanding the great number of hyperopes with poor vision that he examined, Chauvel could find no cause for the poor vision. SULZER.

RAIA (40) discusses asthenopia due to errors of refraction and particularly that due to strain of the recti muscles. He believes with Stevens that difficulty in performing abduction and vertical movements causes reflex asthenopia more frequently than insufficiency of the interni. Exercise, prisms, and tenotomy are recommended in the way of treatment. DANTONE.

In the 4000 eyes reported on by MILES (41), 76 per cent., or 3045, were H., and of those 2384 were simple H., 442 were simple astig. H., and 219 were H. with H. astig. There were 787 M., of which 454 were simple, 182 simple M. astig. and 151 compound M. astig. There were 106 cases of mixed astig. Of the whole number 67½ per cent. were females and the ages ranged from two to eighty years. There were sixteen cases of anerometropia.

BURNETT.

HOWE (42) has demonstrated on *himself* and others the fact that corneal astigmatism can be produced by persistent, strong lid pressure—and has found, in thirty-three *artists* who habitually make the lid pressure in "half-shutting" the eyes at their work, an average astigmatism of .905 D. BURNETT.

THEOBALD (43) reports four additional cases of "subnormal accommodative power," whose chief characteristic is that with an orthophoric condition of the ocular muscles at a distance there is not an exophoria of 3° to 5° for the working distance as should exist when the accommodation is normal. BURNETT.

WHITE (44) has learned from his experience the value of low-grade cylinders as low as 0.25 D in relieving asthenopia, particularly when the axis was off the perpendicular or horizonal or there was anerometropia. One hundred cases are tabulated. BURNETT.

VII.—LIDS.

45. LANDWEHR. On the etiology of chalazion. *Ziegler's Beitr. z. path. Anat. und z. allgem. Pathol.*, xvi., 2, p. 255.

46. MIBELLI. Blepharitis trichophytica. *Ann. di Ottalm.*, xxiii., 5, p. 368.

47. SNELL. Congenital cysts of the eyelids associated with anophthalmus or microphthalmus. *Trans. Ophth. Soc. Un. K.*, xiv., p. 180.

48. DE BOURGON. Unilateral œdema of the lid, the first symptom of a generalized multiform exudative erythema. *Ann. d'Ocul.*, cxii., p. 316.

49. OHLSTRÖM. Congenital bilateral ptosis and immobility of the eyeballs. *Beiträge z. Augenheilk.*, xvi., p. 43.

50. DE SCHWEINITZ. Epithelioma simulating ulcerated Meibomian cyst. *Trans. Amer. Ophthal. Soc.*, 1894.

51. WILMER. A case of melanotic giant-cell alveolar myxoma-sarcoma. *Ibid.*, 1894.

In order to determine whether chalazion has a tuberculous basis, LANDWEHR (45) inoculated the contents of seven chalazia into animals. In no case did tuberculosis develop. Since, however, various observers have found tubercle bacilli in chalazia, we may conclude that only in a small number of cases is the chalazion a tuberculous product, while in the majority it is not.

MIBELLI (46) describes two cases of severe inflammation of the margin of the lid, accompanying trichophytosis on the face, scalp, and neck. Spores of trichophyton were found on the lashes that were pulled out, and the author therefore considers himself justified in calling the disease blepharitis trichophytica. Epilation and antiseptic treatment brought about a cure. DANTONE.

SNELL (47) reports three cases of congenital cysts, two occurring on the lower lid and being associated with anophthalmus, one on the upper lid, associated with microphthalmus. In the latter case there was vomiting when the cyst was punctured. It pulsated synchronously with the anterior fontanelle. Chemical examination of the fluid showed that it was not cerebro-spinal fluid. All three cases were cured, the first two by removing a part of the cyst and the third after three punctures. WERNER.

OHLSTRÖM (49) observed a congenital bilateral ptosis and almost complete immobility of the ball in a boy of fifteen. The accommodation was normal.

DE SCHWEINITZ (50) found a large granular mass protruding from what appeared to be a chalazion of the upper lid. This was excised and the cavity curetted. On examination of the mass it was found to be of an epitheliomatous character. BURNETT.

WILMER'S (51) case was a woman of thirty years who had a growth on the right lower lid which grew very rapidly in size. There was no history of any injury. The conjunctiva was normal. It was removed and measured 12 x 7 *mm* after hardening in alcohol. It had not returned after a year or more had elapsed. It was found to be a melanotic giant-celled myxo-sarcoma. BURNETT.

VIII.—LACHRYMAL APPARATUS.

52. OHLSTRÖM. On extirpation of the tear sac. *Eira*, No. 16, 1894.

53. DE BONO and ALAIMO-MARCHETTI. Iodine injections in the treatment of chronic dacryocystitis. *Arch. di Ottalm.*, ii., 6. p. 195.

54. BAQUIS. Trachoma of the lachrymal gland. xxiii., 3–4, p. 227.

55. SULZER. A contribution to the study of tuberculosis of the lachrymal gland. *Graefe's Archiv*, xl., 5, p. 197.

56. LEPLAT. A case in which death followed probing and injecting the lachrymal canal. *Ann. d'Ocul.*, cxii., p. 328.

OHLSTRÖM (52) gives the following indications for extirpation of the lachrymal sac: 1. Before operations on the ball, when there is not sufficient time for other modes of treatment. 2. When conservative treatment is unsuccessful. 3. In ectasia of the sac, even when the canal is pervious. 4. In tuberculosis of the sac. 5. In obliteration of the canal. 6. With fistula of the sac which resists other forms of treatment. SCHIÖTZ.

53. DE BONO and ALAIMO-MARCHETTI have used with good results injections of iodine in cases of old lachrymal-sac disease, and particularly in those with ectasia of the sac (iodine 0.50, iodide of potassium, 5.00, water 50.00). Care should be taken not to allow the fluid to enter the conjunctival sac. DANTONE.

BAQUIS (54) gives a detailed clinical and histological description of two tumors of the lachrymal gland, removed from a patient having trachoma. From microscopical examination the author makes the diagnosis of trachomatous formation in the lachrymal gland. DANTONE.

SULZER (55) reports a further case of tuberculosis of the lachrymal gland. The gland was removed from a girl of fifteen of tuberculous antecedents.

LEPLAT'S (36) patient suffered from a chronic purulent dacryocystitis which was not improved by injections. A Bowman No. 2 probe was passed easily through the upper two-thirds of the duct, and after this an injection of water was made followed by an injection of 3 % alum solution, which did not pass into the nose. There was no reaction. The following day the procedure was repeated, the alum solution then passing into the naso-pharynx. Shortly after a brown spot was noticed near the lachrymal sac, and the following day the patient showed symptoms of a phlegmon of the face, which grew smaller the second and third day. On the fourth day there was still slight swelling of the sac. The patient complained of pain in the legs. Sudden vomiting. Toward evening, Cheyne-Stokes respiration. Loss of consciousness. T. 39.1. Death two days later. SULZER.

IX.—MUSCLES AND NERVES.

57. SCHWEIGGER. The results of squint operations. *Arch. f. Augenh.*, xxix., p. 165.

58. JOUCK. The various methods of performing squint operations. *Inaug. Dissert.*, Strassburg, 1894.

59. STEVENS. The importance of maintaining the relative amplitude of rotation of the two eyes in operations on the ocular muscles. *Ann. d'Ocul.*, cxii., p. 289.

60. VOSSIUS. A case of traumatic ophthalmoplegia resulting in recovery. *Beiträge zur Augenheilk.*, xvi., p. 34.

61. FRIEDENWALD, N. Traumatic paralysis of the abducens nerve. These ARCHIVES, xxiii., p. 403.

62. HALE, G. W. So-called muscular asthenopia. *Trans. Amer. Ophth. Soc.*, 1894.

According to SCHWEIGGER'S (57) experience, in cases of convergent strabismus with normal binocular vision, the final result of tenotomy of the squinting eye is not sufficient. In periodic squint without diplopia a simple tenotomy should first be done, and if the effect is not sufficient an operation can be done on the other eye. A simple tenotomy in a permanent squint is sufficient only when the average deviation is not over 4 *mm.* A deviation of 5 *mm* can be corrected by tenotomy of both interni, a greater deviation requires advancement. A tenotomy of the externus is successful only in periodic divergent squint. If a squint develops at an age when normal binocular vision has become a habit, diplopia can usually be made out, and the operation restores binocular vision. Usually, however, squint appears at an age when binocular single vision has not become a fixed physiological habit, and even when the operation brings the eyes into such position that normal binocular fusion is possible, it is often not learned.

After describing two cases of traumatic paralysis of the abducens which were operated on with success, JOUCK (58) gives an historical sketch of the squint operation and a description of the different methods of its performance.

In a boy of seventeen, who had received an injury on the left lower lid, VOSSIUS (60) found an almost complete paralysis of all the extrinsic and intrinsic muscles of both sides, and poor vision, all of which was finally completely recovered from. The affection was probably caused by a hemorrhage compressing the nerves of the ocular muscles and the optic nerves.

FRIEDENWALD'S (61) paper forms a continuation of Purtscher's article published in these ARCHIVES. Ten cases in this catagory are noted, and a remarkable case, seen by the author, is added. A man's head was compressed between two railway cars, the force acting transversely on the two parietal bones. Gradually there developed a complete bilateral paralysis of the abducens. That of the left side disappeared in six weeks, while that of the right lasted somewhat longer. There was probably a fracture of the base passing transversely through both temporal bones.

GREEFF.

HALE (62) believes that muscular asthenopia usually has its seat in the central organs, depending upon a disturbance of innervation, or, possibly, in some cases upon a weakness of the power of fusion. In a small per cent. it depends upon the absolute or relative weakness of the adductors or the obliques, or upon their insertion. He depends in treatment entirely upon gymnastic exercise with prisms. BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

63. MITVALSKY. Studies on the osseous tumors of the orbital region. *Arch. d'opht.*, xiv., 10, p. 593.

64. RABINOWITSCH. Echinococcus of the orbit. *Centralbl. f. prakt. Augenh.*, 1894, p. 359.

65. Rauschke, Max. Two cases of inflammatory exophthalmus with fatal ending. *Inaug. Dissert.*, Berlin, 1894.

66. Béclèrc. On thyroidism and its relations to Basedow's disease and hysteria. *Soc. méd. des hopitaux*, Oct., 1894.

67. Clark, E. Impairment of consensual downward movement in right eyelid, with retraction of the lid, and frequent winking in commencing Graves' disease. *Trans. Ophth. Soc. Un. K.*, xiv., p. 244.

68. Herskind. On the surgical treatment and pathogenesis of Basedow's disease. *Bibliotek f. Legar.*, 1894, iv., p. 204.

69. Hjort. Transplantation of skin into the orbit. Norsk, *Mag. f. Laeger.*, No. 7, 1894.

70. Harlan. Three cases of malignant tumor of the orbit. *Trans. Amer. Ophth. Soc.*, 1894.

71. Johnson, W. B. Foreign bodies in the orbital cavity. *Ibid.*, 1894.

Mitvalsky (63) describes the clinical picture and the genesis of osteoma of the frontal sinus with disease of the mucous membrane, leading to retention of contents, distension of the cavity, formation of polypi, and mucous cysts in the tumor and in the orbit, and after suppuration of the cysts, to the formation of fistulæ. His first case was in a girl of fourteen, in whom, after a contusion, there developed a tumor filling the frontal sinus, thinning the anterior layer of the frontal bone, breaking through the inner wall of the orbit, and forming a cyst in the latter. No operation was made at first except the emptying of the cyst, as the ethmoid cells were involved. But when the tumor increased in size, cerebral symptoms being absent, a second operation was undertaken, and an osteoma the size of a fist was removed. After the operation there were wanting the entire cribriform plate of the ethmoid, the lachrymal bone, two-thirds of the upper wall of the orbit, and the entire middle portion of the frontal bone. The tearing away of a portion of the dura adherent to the tumor left the brain bare. Although the nasal cavity communicated with the operative cavity, there was complete recovery with preservation of the eye and its functions. The removal of a small recurring tumor led to permanent recovery. The tumor, weighing 180. gr., of ivory hardness, showed many cavities containing mucous polypi.

In the second case, due to trauma, in a man of thirty, the osteoma, as was seen after its removal, involved the inner two-thirds of the entire roof of the orbit back to the optic foramen. A centimetre from the orbital margin was a polyp of hazel-nut size. A resection was made, after which the growth ceased to increase, and the eye regained its normal position and functions.

The author does not regard the prognosis as being so bad as it was formerly thought to be.

He further reports a case of exostosis of the left orbit in a woman of sixty-five, occupying the entire upper and inner orbital wall, while an ecchondroma sprang from the outer thickened orbital wall, and involved the external rectus. The eyeball was enucleated, but the exostosis could not be removed, and the outcome of the case was unknown. Since the ecchondroma was separated from the exostosis by a layer of periosteum and muscle sheath, the development of the latter from the former was excluded. V. MITTELSTÄDT.

In a girl of seventeen, RABINOWITSCH (64) observed a very rapid development of a left exophthalmus. Outward from and below the protruding ball a tumor could be felt. This was removed with preservation of the ball and proved to be an echinococcus.

RAUSCHKE (65) reports a case of orbital phlegmon in a girl of thirteen, following extraction of a tooth, and one in a man of forty-four, following an abscess in the upper jaw, both having a fatal issue.

HERSKIND (68) gives notes of 42 cases of Basedow's disease from various sources, which were treated by strumectomy or ligature of the thyroid artery, and finds that in 40 % of cases the chief symptoms disappeared completely. SCHIÖTZ.

In two cases of exenteration of the orbit with removal of the entire periorbita and both eyelids, and in a case in which both eyelids, conjunctiva, and ball were removed, HJORT (69) waited until a smooth granulating surface was obtained and then transplanted Thiersch flaps with excellent result. SCHIÖTZ.

70. Case 1.—Epithelial growth evidently beginning in the lachrymal sac in a man of forty years. It was removed, but recurred in three months. Case 2.—Sarcoma, completely filling the orbit, which seemed to start from Tenon's capsule. The eyeball was greatly compressed, but the sclera was sound. Case 3.—Sarcoma of orbit in a child of nine years. It was removed but rapidly recurred and attained a size half as large as the patient's head. Patient died. BURNETT.

All the cases related by JOHNSON (71) were successfully treated except one, which ended fatally from a meningitis caused by a perforation of the anterior lobe of the cerebrum. The size of another would lead to the suspicion that it also had penetrated the brain, but no outward symptoms developed. The histories of four cases in all are reported, and most of the bodies were large. The first was a large twig of cherry tree ; another a piece of pine plank (this penetrated the brain). The third was a piece of pine wood, and in the fourth a mass of grass and bit of weed. In the last case the foreign body struck the back of the eye and produced hemorrhages of the choroid which threatened panophthalmitis, but the eye was finally saved. BURNETT.

XI.—CONJUNCTIVA, CORNEA, SCLERA, AND ANTERIOR CHAMBER.

72. KALT. Treatment of ophthalmia neonatorum. *Arch. d'opht.*, xiv., 12, p. 780.

73. ROHMER. Note on ophthalmia neonatorum. *Annal. d'Ocul.*, cxii., p. 373.

74. PARINAUD. Lachrymal conjunctivitis in the new-born due to the pneumococcus. *Ibid.*, p. 369.

75. SCHANZ. On the etiology of diphtheria. *Deutsche med. Wochenschr.*, 1894, No. 49.

76. SCHIRMER. The clinical picture of conjunctivitis due to the diphtheria bacillus. *Graefe's Archiv*, xl., 5, p. 160.

77. COPPEZ. A case of ocular diphtheria cured by serotherapy. *Annal. d'Ocul.*, cxii., p. 341.

78. HELLGREN. On the mechanical and operative treatment of trachoma. *Hygiea*, 94, 12, p. 569.

79. ANDOGSKY. The operative treatment of xerosis of the eye. *Wratsch*, 1894, No. 43.

80. VAN DUYSE. Two cases of hyaline degeneration of the conjunctiva. *Arch. d'opht.*, xvi., 12, p. 742.

81. LAGRANGE and MAZET. Epithelioma of the bulbar conjunctiva and its propagation into the eyeball. *Ibid.*, p. 768.

82. SANLAY. Note on conjunctival papilloma. *Rec. d'opht.*, 1894, No. 9, p. 543.

83. GASPARINI. Another successful case of transplantation of skin in a case of symblepharon. *Ann. di Ottal.*, xxiii., 34, p. 281.

84. Juda. Subconjunctival cysticercus. *Med. Weekblad.*, 1894.

85. Bocchi. On the recurrence of pterygium. *Arch. di Ottal.*, ii., 3–4, p. 120.

Kalt (72) believes that careful cleansing and disinfection of the conjunctival sac are all that is necessary in the treatment of blennorrhœa. He places a funnel of ebony between the lids, and through a tube connected with it two quarts of solution of permanganate of potassium are passed into the conjunctival sac twice daily or oftener. v. Mittelstädt.

Rohmer (73) reports several cases which suggest facts in the nosology and therapy of ophthalmia neonatorum that are too often forgotten. At times severe cases recover without treatment, at others the cornea is perforated in spite of the most careful treatment. Sulzer.

Parinaud (74) calls attention to the fact that obstetricians and midwives often see a benign but tedious conjunctival inflammation of the new-born that is rarely observed by the oculist. In most of these cases there is obstruction in the lachrymal duct, and there is rhinitis present. The affection is usually cured in three or four weeks with the use of boric acid washes, or even without treatment. The pneumococcus is found in the secretion in every case, the gonococcus and Weeks' bacillus never. Sulzer.

From the study of a case of infantile xerosis with keratomalacia, Schanz (75) concludes that the xerosis bacillus is a Löffler's diphtheria bacillus of diminished virulence.

In 8 cases of croupous conjunctivitis Schirmer (76) found the Löffler bacillus always present.

Coppez (77) believes that injections of antitoxin exert a favorable influence on diphtheritic conjunctivitis. In one case, in a girl of twelve, in which the local use of oleum cadini did not affect a diphtheritic conjunctivitis with an adherent membrane, a single injection caused a rapid improvement and the dissolution of the diphtheritic exudation. Bacteriological examination revealed many diphtheria bacilli and a few streptococci. Sulzer.

Of the various mechanical and operative methods of treatment, Hellgren (78) prefers expression. He reports 42 cases operated on in this way. He frequently used Knapp's roller-forceps with smooth rollers. The results were good on the whole although keratitis developed after the operation in 4 cases, and in one lasted a long time and reduced vision to $\frac{1}{6}$.

(Corneal ulcerations, which are mentioned by several authors and which I have never met with, probably have occurred in cases of trachoma complicated with an infective conjunctivitis. Smooth cylinders, in my experience, have been less efficient than creased ones.—H. K.)

AUDOGSKY (79) operated on 5 eyes of 13 patients with trachomatous xerophthalmus after Rudin's method, which consists in a narrowing of the palpebeal fissure, with the expectation that by diminishing the extent of the uncovered dry surface, the secretion from the surface and glands of the conjunctiva will be more apt to keep the cornea moist. HIRSCHMANN.

LAGRANGE (81) describes another case of epithelioma observed with MAZET. The tumor, having the size of a franc piece, occupied the inner-upper sclero-corneal region of the right eye of a woman of sixty-five, and extended inward in a cone-shaped process between the ciliary body and the sclera. v. MITTELSTÄDT.

GASPARINI (83) reports the successful transplantation of two flaps of skin from the forearm to cure a symblepharon. The scar of the latter, which followed the removal of an epithelioma, extended to the corneal margin and had caused glaucoma. The pain demanded operative interference.

The operation not only restored the mobility of the ball, but led to complete disappearance of the glaucomatous symptoms and to the recovery of useful vision. DANTONE.

JUDA (84) removed a cysticercus from beneath the conjunctiva of a child of six. WESTHOFF.

BOCCHI (85) describes the histological process that takes place in the recurrence of pterygium. This is more frequent when the entire pterygium is removed and the wound cauterized, than when the removal is only partial and the conjunctiva is sutured.

DANTONE.

86. BJERRUM. Subconjunctival injections. *Med. Aarsskrift*, 1894.

87. SANTO DOMINGO. On congenital opacity of the cornea. *Inaug. Dissert.*, Berlin, 1894.

88. SNELLEN. Epithelioma of the cornea. *Med. Weekblad.*, 1894.

89. DE WECKER. Treatment of injuries of the cornea by occlusion with conjunctiva. *Annal. d'Ocul.*, cxii., p. 293.

90. KYLE. A case of gumma of the sclerotic. *Amer. Journ. of Ophth.*, Nov., 1894.

BJERRUM (86) reports an obstinate case of kerato-iritis treated without result, in which 3 subconjunctival injections of a solution of salicylate of soda cured the disease completely. He had often used this remedy and believed that he had seen favorable results while he had not seen results from the injection of sublimate.

SCHIÖTZ.

SNELLEN (88) reports a case in which an epithelioma of the cornea developed after a pterygium had been removed twice. The ball was enucleated. WESTHOFF.

DE WECKER (89) in cases of corneal injury advises covering the entire cornea with a conjunctival flap. The conjunctiva is loosened about the cornea, as in the abscission of staphyloma, and drawn together over the cornea by a tobacco-pouch suture or by four to six sutures applied in a vertical line. SULZER.

The case which KYLE (90) reports is evidently one of what is known as gumma of the ciliary body, in a man of twenty-two, who had the initial lesion of syphilis sixteen months before.

BURNETT.

Sections XII.–XXI. Reviewed by DR. P. SILEX.

XII.—IRIS.

91. PFAMMÜLLER. A contribution to the colobomas of the eye. *Inaug. Dissert.*, Giessen, 1894.

92. MOEGELE. On iritis with nodular formations. *Inaug. Dissert.*, Tübingen.

93. MALGAT. Spontaneous mydriasis. Recovery after strychnine injections. *Rec. d'opht.*, 1894, p. 714.

94. HOWE, L. Three cases of so-called ectropion uveæ. *Trans. Amer. Ophth. Soc.*

95. OLIVER. Some additional studies upon the clinical value of repeated careful correction of manifest refractive errors in plastic iritis. *Ibid.*, 1894.

96. DE BECK. A family history of irideremia and coloboma. Cataract operation on two members. *Ibid.*, 1894.

PFAMMÜLLER (91) reports on 16 patients with 18 colobomatous eyes, seen in Giessen in the last four years. There were 3 colobomas of the optic nerve, 1 of the macula, 1 of the lens, 10 of the iris, and 10 of the lids. The various theories as to the origin of coloboma are discussed.

MOEGELE (92) believes that two of his cases of iritis serosa with grayish-red and white nodules in the iris are identical with those described by Liebrecht and Vossius, and called by the latter iritis with nodular new formations resembling tubercles. Tuberculosis could be ruled out.

MALGAT'S (93) patient, a healthy woman of fifty, after great excitement, had an attack of headache lasting through the night. The following day the sight of the left eye was affected. Ten days after, the left pupil was dilated ad maximum, the eye being otherwise normal. Injections of strychnine twice a week cured the affection in a month. SULZER.

HOWE (94) reports three cases of pigmentary excrescences projecting from the edge of the pupil into the anterior chamber, some of which are pedunculated. In two there was myopia of high degree. He thinks all such cases should be classed under papillomata iridis. BURNETT.

By a number of carefully conducted experiments OLIVER (95) has shown that the so-called " spastic accommodation " in plastic iritis is not due to a forward displacement of the lens nor to increase in the aqueous or vitreous humors. BURNETT.

DE BECK'S (96) cases were in two brothers who were affected with double cataract, the one with irideremia and the other with a downward coloboma of the iris. The former was forty-two years old, the latter thirty-six, and vision had never been good in either case. The cataracts had been developing for two or three years. The four cataracts were extracted, but there was excessive reaction in all—and one fairly good eye was saved for each one. The author infers that in these cases of anomalies of the uveal tract there is a liability to serious reaction after operations. In regard to these defects, it is interesting to note that in the first generation there were two brothers, one with irideremia and the other with coloboma; in the second generation the brother with irideremia has one son with irideremia and another with coloboma, while the brother with coloboma has a son with irideremia; in the third generation the son with irideremia has three children (sons) with irideremia; the son with coloboma has a daughter with irideremia. BURNETT.

XIII.—CHOROID.

97. Ginsberg. Exudative choroiditis and multiple pedunculated polypoid granulation-tumors of the choroid as pseudoglioma. *Centralbl. f. Augenheilk.*, xviii., p. 322.

98. Wiegmann. A case of choroiditis senilis maculata. *Klin. Monatsbl. f. Augenheilk.*, xxxii., p. 457.

99. Fage. Rupture of the choroid. *Soc. d'opht. de Paris*, Nov. 6, 1894.

100. Terson. Early gummata of the ciliary body. *Arch. génér. de méd.*, Oct., 1894.

101. Berger. Colloid degeneration of the endothelium of the posterior chamber. *Arch. d'opht.*, xiv., 2, p. 677.

102. Theobald. A case of panophthalmitis suppurativa following discission of a capsular opacity. *Trans. Amer. Ophth. Soc.*, 1894.

103. De Schweinitz. Colloid disease in the macular region analogous in appearance to the so-called disease in the nerve head. *Trans. Amer. Ophth. Soc.*, 1894.

In Ginsberg's (97) case, the eye showed the clinical picture of glioma and was enucleated. Microscopically there was found an exudative choroiditis which had led to detachment of the retina and atrophy of the choroid, with great proliferation of the pigment epithelium, and had produced a multiple pedunculated tumor arising from the choroid and projecting free into the interior of the ball. There was no history of syphilis. Granulation tumors have been described by Leber, Wedl-Bock, and Pagenstecher-Genth.

Terson (100) calls attention to the fact that a gumma of the ciliary body coming on in the course of a specific granuloma iritis is easily diagnosticated, while a primary early gumma of the ciliary body is usually mistaken for neoplasm and the eye enucleated. Primary gumma of the ciliary body is often an early tertiary manifestation of syphilis. In the beginning the symptoms are not characteristic, being pain and diminution of vision. Following this latent period comes swelling of the sclera in the ciliary region, and later the iris becomes infiltrated, and cauliform excrescences form at its base, the latter symptom by itself justifying a positive diagnosis. Sulzer.

Berger's (101) case was one of benign tuberculosis of the uveal tract. He found a thickening of the membrana limitans iridis and at points a swelling of the pigment epithelium which was separated from the membrana limitans by a layer of fluid, and also showed cavities filled with fluid, among the cells. On the posterior surface of the membrana limitans were numerous hemispherical, homogeneous, yellow formations having the size and histological characteristics of hyaloid formations on the membrane of Descemet. There were also many of the colloid balls in the anterior chamber. Similar changes were found in the endothelium on the lamina vitrea of the pars ciliaris retinæ in an eye with iridocyclitis, and in an atrophied ball with preservation of the anterior chamber. The author believes that the endothelium of the posterior surface of the iris has the same significance as that of Descemet's membrane and the cells of the anterior capsule of the lens, and that a disturbance in these cells may also lead to nutritive disturbances in the neighborhood.

v. MITTELSTÄDT.

THEOBALD'S (102) patient was a negro boy of fourteen years, on whom he had operated several times on both eyes for cataract by discission. To remove a remaining capsular opacity in the right he needled it after the usual aseptic precautions. In seventy-four hours the cornea was necrotic and the ball went on to atrophy. —Knapp said in the discussion that in more than 1500 discissions for secondary cataract he had not had a single case of that kind.

BURNETT.

DE SCHWEINITZ (103) reports two cases with a fine chromolithograph of colloid degeneration at the macula of both eyes—with unimpaired vision. BURNETT.

XIV.—GLAUCOMA.

104. ANGELUCCI. On disturbances in the vascular apparatus that are met with in patients with hydrophthalmus congenital or acquired. *Arch. di Ottal.*, ii., 1–2. p. 24.

105. OLIVER. A clinical and microscopical study of two cases of glaucoma associated with intraocular hemorrhages. *Trans. Amer. Ophth. Soc.*, 1894.

ANGELUCCI (104) is led by experiment to believe that hydrophthalmus is caused by vaso-motor changes in the uveal tract, which in their turn are to be referred to disturbances in conduction in the region of the trigeminus and cervical sympathetic.

Under these trophic influences, damage is done to the choroid, iris, and cornea while the retina does not suffer. DANTONE.

These two cases, whose history OLIVER (105) has followed out serve to illustrate the two opposite issues that may occur in glaucoma which seems to be secondary to retinal hemorrhages. In one the eye went on to destruction, in the other some vision was saved, even though a lens which had become cataractous had to be extracted. Iridectomy was done in both cases. In the first (unsuccessful) case, there was a perivasculitis associated with grave central nervous disease. In the other there is a probability that the vascular walls were not so much diseased, and the author agrees that there may be still hope for cases of glaucoma following hemorrhage under proper hygienic care. BURNETT.

XV.—SYMPATHETIC OPHTHALMIA.

106. BJERRUM. On the pathogenesis of sympathetic ophthalmia. *Med. Aarsskrift*, Copenhagen, 1894.

107. LANG. Sympathetic inflammation beginning ten days after a penetrating wound just behind the sclero-corneal junction. *Trans. Ophth. Soc. Un. K.*, xiv., p. 218.

108. HOBBY. Are gun-cap wounds of the eye without danger? *Annals of Ophth. and Otol.*, Oct., 1894.

HOBBY'S (108) experience has been that fragments of percussion caps (copper) are liable to produce sympathetic ophthalmia, especially when the wound is in the ciliary region. He gives five cases in support of this, and says that there are twenty cases in the College for the Blind in Iowa who lost the second eye from sympathy with the other, which had been wounded by a fragment of percussion cap. BURNETT.

XVI.—LENS.

109. KNAPP. On glaucoma after discission and its treatment. *Arch. f. Augenheilk.*, xxx., p. 1.

110. STEPHENSON. Coloboma of each lens. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 211.

111. CHRISTEN. Three cases of congenital coloboma of the lens. *Arch. f. Augenheilk.*, xxix., p. 233.

112. PAGENSTECHER. Practical points in cataract extraction. *Klin. Monatsbl. f. Augenheilk.*, xxxii., p. 439.

113. SNELL. Case presenting unusual appearances after extraction of cataract; simulating a cyst, really due to a distended capsule. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 135.

114. KALT. Corneal suture after extraction of cataract. These ARCHIVES and *Arch. d'opht.*, xiv., p. 10.

115. CLARK, C. F. A case of binocular coloboma of the lens with accommodative power retained. *Trans. Amer. Ophth. Soc.*, 1894.

Altogether, KNAPP (109) has seen 15 more or less severe attacks of glaucoma follow discission of secondary cataract. These attacks came on particularly after extensive splitting of the capsule and deep penetration of the knife into the vitreous, and also when considerable masses of cortex had been left. Yet probably the swelling is not so much the cause of the glaucoma as is traction on the ciliary processes. This traumatic glaucoma may be cured spontaneously, or it may lead to blindness. Some cases recover through the use of eserine and hypodermic injections of morphine, while others require iridectomy. Posterior sclerotomy had not been successful; anterior sclerotomy had not been tried. (The reviewer cured two cases of this sort permanently by puncturing the anterior chamber.)

CHRISTEN (111) describes 5 congenital colobomas of the lens seen in 3 patients, only 46 cases in 38 individuals having been previously reported. The affection is usually bilateral. Four times the coloboma was downward, once upward and outward. In 2 instances the capsule was normal notwithstanding the defect in the lens, which speaks for the independent development of lens and lens capsule. There was also coloboma of the iris in 4 of the eyes. Vision was fair in 4 and poor in 1 eye.

PAGENSTECHER (112) advises beginning operators not to be led astray and confused by the new modifications suggested every day in the extraction of cataract, but says that they should choose one operation and become proficient in it. The beginner should do the operation with iridectomy, and discission should be done with a thin conical needle passed through the sclero-corneal margin.

In SNELL'S (113) case the lens was extracted, some vitreous escaping. $V = \frac{6}{9}$. Six or seven years later the patient noticed a small white body in the anterior chamber. There was a pear-shaped cyst in the lower part of the eye connected by a thin thread with the iris, and this cyst proved to be formed of lens capsule. WERNER.

CLARK (115) describes a case where there was a defect in both lenses below the edge, forming a horizontal line. The eyes were myopic, but possessed good accommodation, and the total astigmatism was much less than the corneal when the eye was not under the influence of a mydriatic, but was nearly the same when it was atropinized. BURNETT.

XVII.—VITREOUS.

116. PRESSEL. A case of recurrent hemorrhage into the vitreous in consequence of menstrual disturbances. *Inaug. Dissert.*, Würzburg, 1894.

117. v. HIPPEL, E. On recurrent intraocular hemorrhages caused by a tumor. *Graefe's Archiv*, xl., 4, p. 260.

118. BATTEN. Persistent hyaloid artery branching in the vitreous. *Trans. Ophth. Soc. Unit. King.*, xiv., p. 209.

PRESSEL (116) reports a case of hemorrhage into the vitreous occurring fifteen times within a few months in a girl of sixteen who had never menstruated. It was supposed that the hemorrhages were due to an hereditary specific vascular affection which caused the terminal arteries to become brittle.

v. HIPPEL (117) reports two cases of recurrent intraocular hemorrhage due to a tumor. The anterior chamber became filled with connective tissue, and there were marked inflammatory changes in the interior of the eye. The ball did not perforate. From the fibrin of the blood there had formed a homogeneous, colloid-like substance that gave the characteristic reaction for amyloid.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

119. GUIFINKEL. A case of spontaneous cure of detachment of the retina. *Wjest. Ophth.*, No. 6, 1894.

120. UHTHOFF. Demonstration of a case of reattachment of the retina, and a case of tuberculosis of the upper lid. *Berliner klin. Wochenschr.*, No. 31, 1894.

121. SCHEFFELS. Cure of myopic detachment of the retina. *Festschr. z. 50-jähr. Jubiläums d. Vereins d. Aerzte d. Reg. Bez. Düsseldorf*, p. 280. Bergmann, Wiesbaden, 1894.

122. TSCHMEMOLASSOW. Hemorrhage in the retina in pernicious anæmia, due to tapeworm. *Petersb. med. Wochenschr.*, 1894, p. 50.

123. CRAMER and SCHULTZE. On pseudo-glioma of the retina. *Arch. f. Augenheilk.*, xxix., p. 288.

124. BERGER. Syphilitic retinitis with albuminuria. *Rev. génér. d'opht.*, xiii., p. 490.

125. KATAYAMA and OKAMOTO. Studies on amaurosis and amblyopia, caused by the malefern. *Vierteljahresschr. f. gerichtl. Med. u. öffentl. Sanitätswesen*, 1894, suppl.

126. DE HAAS. On epidemic hemeralopia. *Tydschr. voor Geneeskunde*, ii., p. 999, 1894.

127. SIMON. On the origin of the so-called fatigue contraction of the visual field. *Graefe's Archiv*, xl., 4, p. 276.

128. BOUVIN. Embolism of the central artery of the retina. *Med. Weekblad*, 1894.

129. WAGENMANN. Disappearance of medullated nerve fibres in the retina in consequence of tabic optic-nerve atrophy. *Graefe's Archiv*, xv., 4, p. 257.

130. BULL. Recent experiences in the treatment of detached retina, with a detailed report of thirty-eight cases. *Trans. Amer. Oph. Soc.*, 1894.

131. WURDEMANN. Report of a case of thrombo-phlebitis of the central retinal vessels, with necropsy. These ARCHIVES, xxiii., p. 445.

132. DE SCHWEINITZ. Concerning monocular diplopia, with cases. *Ibid.*, 1894.

133. HEYL. Rupture of the lymph sheath of a retinal vein. *Ibid.*, 1894.

In GUIFINKEL'S (119) case there was detachment of the retina in an eye, with myopia, sclerectasia, and atrophic changes in the fundus, on which an iridectomy had been done for exclusion of the pupil. Treatment was of no benefit, but eight months later the retina had become spontaneously reattached, with restoration of the previous vision, and in three years there has been no change. HIRSCHMANN.

SCHEFFEL'S (121) patient with detachment of the retina was cured in nine months by rest in bed and alterative and diaphoretic treatment. Cases treated by Schöler's method did not do well.

Pagenstecher in one case made multiple punctures through sclera, choroid, and retina, in the hope of exciting an adhesive chorioretinitis, and obtained a permanent result.

Tschmemolassow (122) gives a preliminary report on four cases of pernicious anæmia, caused by the bothriocephalus latus, that were speedily cured by removal of the parasite. In all four cases there was retinal hemorrhage, varying in size from the punctate to such as were as large as the disc. These hemorrhages are due to the anæmia, and not to the parasite directly.

Cramer aud Schultze (123) give a clinical and anatomical description of a case of pseudo-glioma in a girl of fifteen. The clinical picture was that of glioma, excepting the age of the patient. The symptoms diagnostic of pseudo-glioma were mostly wanting. The enucleated ball was 18–19 *mm* in all diameters. The retina was totally detached, and the vitreous chamber was filled with a whitish exudation. The ciliary body was atrophic. There was diffuse fibrous degeneration of the entire choroid with fresh inflammatory foci, and near the optic nerve numerous pigmented spots with giant-cells, such as are found about foreign bodies, and cholesterin crystals. Pseudo-glioma is only a negative diagnosis, glioma being mostly confounded with purulent choroiditis, due to infectious diseases, and particularly often, in the opinion of the authors, to measles.

A patient, who had taken 3.00 of malefern daily for twelve days for anchylostomum duodenale, led Katayama and Okamoto (125) to study the toxic effect of this drug on vision. The amblyopia, like tobacco amblyopia, seems to affect the weak and anæmic most readily. Three to ten grammes a day, taken for several days, causes a general poisoning or amaurosis in man, and a much smaller quantity has a similar effect on dogs.

In opposition to Walter, who believes that acute hemeralopia is dependent upon a miasma, de Haas (126) believes that in every case there has been a blinding of the eyes by strong light. The disease occurs only in individuals who work in the open air. Direct sunlight is the cause and the cases are cured without other treatment if the eyes are protected from the light. The xerosis of the conjunctiva that often accompanies hemeralopia he considers the first symptom of the burning and drying of the conjunctiva from the action of the sun and the wind. Westhoff.

It is now commonly recognized that symptoms of fatigue appear in the course of perimetric tests, both in the healthy and the diseased eye, but their explanation is still in doubt. Schiele locates the fatigue in the cortex of the occipital lobe, Wilbrand in the retina, and others in the consciousness. SIMON (127) has now made a number of experiments and has found, among other results, that the symptoms of fatigue do not come on when large test objects are used, and when the test objects are carried along with a twitching motion.

BOUVIN'S (128) patient complained of having had blurred vision for two hours, $V = \frac{3}{60}$ E. In the retina was a white exudation, and in two arteries the blood column was interrupted. The eye was massaged twice and the following day $V = \frac{6}{24}$, and the exudation had almost disappeared. The massage was continued and the visual acuteness became normal, with a small defect in the field inwardly. WESTHOFF.

WAGENMANN (129) describes one of the rare cases of tabic atrophy that begin with a central scotoma. Later a defect appeared upward and inward, and at the same time a group of medullated nerve fibres occupying the corresponding position in the fundus lost their medulla. In this case, therefore, it must be supposed that the destructive process which can affect the medulla and the axis-cylinder simultaneously, passed into the eye relatively early, the atrophy therefore not being a descending one.

From a study of these thirty-eight cases, BULL (130) concludes that there has been no better means of dealing with detached retina than the old methods of rest in bed, atropine, a bandage, and the internal administration of some drug that may induce absorption of the subretinal fluid. For this latter he does not consider pilocarpine the best, but prefers small doses of bicarb. of soda and iodide of potas. Puncture can give only a temporary relief, and severing of bands in the vitreous should be made only when they are non-vascular. The methods of Schoeler he condemns entirely. BURNETT.

WÜRDEMANN (131) examined a boy of eight, whose eye had become blind four days before in consequence of measles. There was stoppage of the central vein, dilatation and tortuosity of the retinal veins, striped retinal hemorrhages, and œdema of the macular region. The arteries were very small. The picture resembled that of neuro-retinitis, but a diagnosis of thrombosis of the central veins was made, and the eye having been enucleated later for secondary glaucoma, microscopic examination confirmed the diagnosis. GREEFF.

In de Schweinitz's (132) first case a man received an injury to the head and suffered from very pronounced nervous symptoms referable to a central lesion. He had diplopia in the left eye alone. The true image is vertical, the false image in front of it, and inclined towards the left. No optical defect or opacity in the media ; fundus normal. In the second, an alcoholic, with central nerve lesions, astigmatism, old iritis, capsular opacities, and cerebral choroiditis, had vertical diplepia more particularly in the left eye. Burnett.

Heyl (133) reports and pictures a rupture of the lymph sheath of a retinal vein. The vein was tortuous and projected at places into the vitreous. In close proximity to the ruptured sheath and attached to the optic disc were white plaques evidently of a connective-tissue character, and which the author thinks are related to the lymph as effects to a cause. He considers them as organization of the leucocytes which escaped from the ruptured sheath. Burnett.

XIX.—OPTIC NERVE.

134. Elschnig. On so-called choked disc. *Wien. klin. Wochenschr.*, 1894, No. 51.

135. Bjerrum. On optic atrophy in syphilitic patients. *Med. Aarsskr.*, Copenhagen, 1894.

136. Burnett. Tumor of the intervaginal space of the optic-nerve sheath. *Trans. Amer. Ophth. Soc.*, 1894.

Elschnig (134) remarks that in the last decades the mechanical theory of the production of choked disc has gradually given place to the Leber-Deutschmann inflammatory theory. Elschnig believes choked disc to be a form of intraocular neuritis, characterized by increase in the volume, prominence, and area of the disc, caused by micro-organisms and their products.

In order to decide whether an optic-nerve atrophy is primary or secondary, Bjerrum (135) lays down the following rules : 1. Unilateral affection indicates secondary atrophy ; in primary both nerves are always affected. 2. Hemianopic defects in the field are excessively rare in primary atrophy. 3. Improvement in function, even transitory, is argument against primary atrophy. Schiötz.

Burnett's (136) case was a boy of seven years whose left eye was noticed to protrude slightly three years before he saw him.

The exophthalmos slowly increased, but vision was lost very early. There was no pain ; nerve white and showed evidences of a past papillitis. Movements of globe limited equally in all directions. Eye enucleated and a mass of material attached to the nerve was taken from the orbit afterwards. An examination of sections of the nerves and of the mass showed the same appearances that are usually found in tumors of the intervaginal space. In some portions it was myxomatous, in others spindle-celled, in others fibrous, in others like endothelioma. He regards these growths not as pathological, or at least not malignant, but more of an embryonal character.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

137. HILLMANNS. On injuries of the eye. *Arch. f. Augenheilk.*, xxx., 1., p. 29.

138. KNABE. A contribution to the statistics and clinical histories of eye injuries. *Inaug. Dissert.*, Hälle, 1895.

139. SPECHTENHAUSER. On foreign bodies in the eye. *Wien. klin. Wochenschr.*, 1894, No. 43.

140. WAGENMANN. Report on the extraction of a splinter of glass from the anterior chamber, with remarks on the inflammation caused by foreign bodies. *Graefe's Archiv*, xl., 5, p. 180.

141. FROMAGET. Tetanus following traumatism of the eye and its adnexa. Ocular symptoms of tetanus. *Arch. d'opht.*, xiv., 77, p. 657.

142. KNAPP. Two recent magnet operations, one an ideal success, the other a total failure ; with remarks. *Trans. Amer. Ophth. Soc.*, 1894.

HILLMANNS (137) reports on 175 cases of eye injury seen within a year at the Bonn Clinic. Serpiginous ulcer is treated by Saemisch's method.

KNABE (138) reports on 205 cases of eye injury seen within two years in the Halle Clinic. Fine silk sutures are used in cases of gaping wounds of the cornea or sclera.

One of SPECHTENHAUSER'S (139) cases was the spontaneous extrusion through the sclera of a bit of gun-cap that had been in the eye seven years, and one in which an iron splinter had remained in the fundus twenty-eight years. Only three other cases of spontaneous extrusion of a foreign body through the sclera have been described.

Wagenmann (140) had under observation for a year a case in which a splinter of glass had entered the anterior chamber without injuring the iris or the lens. In the course of time a corneal opacity appeared, such as Leber produced experimentally in rabbits. This was no doubt due to chemical influences, as the foreign body was immobile and could not have caused trauma, and the course of the affection and inoculations after removal of the foreign body showed that it was aseptic. The opacity cleared up after the removal.

Seven days after the loss of the left eye from injury by a rocket, Fromaget's (141) patient lost the mobility of the healthy eye and developed a trismus. The pupil was of normal size, but reacted only to accommodation. Sight and accommodation normal. Mobility of the lids normal. The injured eye was removed, and the following day tetanus developed, from which the patient died five days later. The author collates a number of observations of tetanus after injury of the eyeball, lids, and orbital margin, those of the orbital margin having proved most dangerous in this respect. A more detailed account is given of the cases in which spasm and paralysis of the facial and ocular muscles have occurred. These symptoms, the author thinks, are due to infection, like the post-diphtheritic paralyses. In most cases the pupils were small and unresponsive.

In Knapp's (142) first case the bit of steel entered at the outer edge of the cornea. The foreign body was seen in the lower part of the retina, some blood in vitreous, and an air bubble in upper part. $V=\frac{20}{20}$. Primary operation refused, but two days later when the eye showed signs of beginning inflammation he returned and the bit of steel was extracted by a Hirschberg magnet through an incision between the external and inferior recti muscles. There was no escape of vitreous, the wound healed without reaction, and V was $\frac{22}{20}$. In the second case a piece of iron penetrated the eye. Its interior was disorganized. The foreign body was not found with the magnet nor after enucleation. Burnett.

XXI.—OCULAR AFFECTIONS IN GENERAL DISEASES.

143. Siegrist. Contributions to the knowledge of the nature and location of ophthalmic migraine. *Thèse de Lausanne*, 1894. *Annales suisse des sciences medicales*, 1894.

144. Peretti. Traumatic bitemporal hemianopia with remarks

on hemianopic pupillary reaction. *Festschrift z. Feier d.* 50 *jährigen Jubiläums d. Vereins d. Aerzte Reg.'Bez. Düsseldorf*, 94.

145. GUILLERY. On latent ocular muscle disturbances in tabes. *Arch. f. Augenheilk.*, xxix., p. 361.

146. HESSBERG. Ocular affections following influenza. *Festschr., Düsseldorf*, 1894.

147. ADAMÜK. Ocular affections after typhoid processes. *Petersb. med. Wochenschr.*, 1894, 38, 39.

148. EDINGER. A new theory of the cause of some nervous diseases, particularly neuritis and tabes. *Volkmann's Sammlung klin. Vorträge*, 94, No. 106.

149. DEGENKOLB. Experiments on the influence of some medicaments on the power of the eye to recognize slight differences in light intensity. *Inaug. Dissert*, Tübingen, 1894.

150. BAAS. On an ophthalmia herpetica. *Graefe's Archiv*, xv., 5, p. 212.

151. WEYMANN. A case of quadrisectorial homonymous hemianopsia. *Amer. Journ. of Oph.*, Oct., 1894.

In SIEGRIST'S (143) cases there were vasomotor disturbances, those localized in the dura being capable of causing the migraine and those in the occipital cortex the scintillating scotoma. The vasomotor disturbance is probably a spasm of the vessels of one hemisphere causing an increased excitability. The causes are refractive errors, unusual strain on the eye, or a congenital disposition, and the exciting causes are hunger, fright, weakness of the heart, etc.

PERETTI (144) reports a case of fracture at the base of the skull with bitemporal hemianopsia. At first there was no difference in the pupillary reaction when the two halves of the retina were illuminated separately, but after the left eye had become amaurotic pupillary reaction followed only when the temporal half of the retina was strongly illuminated. From this he concluded that so long as the centre of the retina functionates, the diffusion and reflection of light within the eye affects the pupillary reaction so that the presence or absence of hemianopic pupillary reaction is not a safe diagnostic sign in most cases of hemianopsia.

GUILLERY (145) states that disturbances of the ocular muscles, particularly such as cause transitory diplopia, indicate nervous disease, which is rarely recognized early as the oculist is only consulted when the symptoms are marked. Often there is no spontaneous diplopia and the patient complains of blurred or flickering vision only, and in such cases the diagnosis of muscular disturbance must be made by means of some of the tests for heterophoria.

Hessberg (146) shows by clinical histories that all parts of the eye may be affected in influenza, but without other characteristic symptoms than are found in the ocular affections with any infectious disease.

Edinger (147) offers a function theory to explain nervous diseases. Function is a wearing out of the nervous elements, for which in normal conditions metabolism furnishes a restoration. When, however, the function is overstrained or in case injury or disease affects the restoration, the functionating nervous elements are destroyed and the intervening tissue proliferates.

This hypothesis explains not only the disturbances in the eye in tabes, but also the pupillary symptoms, and, according to it, the fibres governing the light reflex must suffer before those governing the accommodation. The levator, abducens, and internus are therefore the earliest and most frequently affected. When the wearing out is not excessive, the affected cell may be restored, and thus the ocular paralysis may disappear and recur. The argument is ingenious, but purely hypothetical.

Degenkolb's (149) experiments were carefully carried out with the aid of Hüfner's spectro-photometer, and confirmed Dreser's finding in regard to the action of strychnin. 0.003 injected into the cheek notably increased the power of the author to distinguish differences in luminous intensity. The action of tea and coffee was doubtful, but alcohol always reduced the perception of differences in luminous intensity.

Baas's (150) extracts from the literature show that our knowledge of the connection between eye and liver affections is still incomplete, and two cases that he reports are of great interest. Both were cases of cirrhosis of the liver with icterus in young individuals. Soon after the development of the affection hemeralopia was noticed which persisted while the icterus lasted, and then disappeared. All the vessels of the eye showed endarteritis or endophlebitis leading to circulatory disturbances, with emigration of cells which were in part diffusely scattered and in part gathered into groups.

In WEYMANN'S (151) case, a man, who was evidently a syphilitic, had a paralysis of the right side, and other symptoms pointing to a severe central lesion. The peculiarity of the eye trouble was sectorial defects in the visual fields of both eyes. They were homonymous—that is, down and out and up and in in the right, and down and in and up and out in the left, and the points of the angular sectors came together at the points of fixation. The nerve-head seemed pale, but the vision was fairly good ($\frac{20}{40}$). His general condition improved under heavy doses of iodides, but the eye symptoms remained essentially the same.

BURNETT.

Obituary.

Dr. M. Landesberg died at Florence, March 4, 1895. He practised in New York for a number of years, and published many valuable papers, among which we mention: On Keratitis Bullosa, these Archives, vol. vi., p. 135–169; On the Pathology of the Sympathetic Nerve, *Ibid.*, vol. vii., p. 408, 1878; The Use of Eserine in Glaucoma, *Ibid.*, vol. viii., p. 264; Sarcoma of Choroid in Children, *Ibid.*, vol. viii., p. 345.

BOOK NOTICE.

Skiascopy and its Practical Application to the Study of Refraction. By Edward Jackson, A.M., M.D. The Edwards & Docker Co., Philadelphia. 109 octavo pages, with many illustrations. Price, $1.00.

This little book gives a very complete and serviceable presentation of the theory of the shadow-test and its practical applications. To many it will seem too diffuse, but in reality it contains but little superfluous matter, at least for those who have but an imperfect previous knowledge of this useful method of examination. Even one who has somewhat extensive experience in the latter will find information and many useful hints, some of which may be new to him. To all interested in the subject we can cordially commend Dr. Jackson's monograph. A. D.

Contents of the last numbers of the Archiv für Augenheilkunde (German edition of these Archives).

Vol. XXX., No. 4. Issued in May, 1895.

A.—ORIGINAL PAPERS.

XXIII. O. Walter. Report and Experiences of an Itinerant Cataract Operator in the Latter Part of the Nineteenth Century.

XXIV. Rudolf Denig. Anatomical Description of Spontaneous Connective-Tissue Formation in the Vitreous (so-called retinitis proliferans).

XXV. ALEXANDER TEPLJASCHIN. On the Pathological Anatomy of the Intrauterine Inflammations of the Eye and Particularly the Congenital Opacities of the Cornea (two plates).

B.—REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FOURTH QUARTER OF THE YEAR 1894. (Translated in this number.)

C.—MISCELLANEOUS.

Vol. XXXI., No. 1. Issued in June, 1895.

A.—ORIGINAL PAPERS.

I. MOCHEK. Herpes Zoster of the Iris in the Course of Herpes Zoster Fontalis.

II. GIACOMO ROMBOLOTTI. Clinical and Anatomical Contribution to the Simple or Serous Cysts of the Conjunctiva (one plate).

III. O. STUELP. A Case of Traumatic Pulsating Exophthalmus with Autopsy, with one illustration in the text.

IV. P. V. RICHTER. On Intermittent Exophthalmus.

V. RICHARD HILBERT. On So-Called Double Sensations.

VI. E. ASMUS. On the Accurate Localization of Large Iron and Steel Foreign Bodies in the Eye by Means of the Sideroscope.

VII C. MELLINGER and D. BOSSALINO. Experimental Study on the Diffusion of Fluids Injected Subconjunctivally.

VIII. SEGGEL. A Further Case of Unilateral Reflex Iridoplegia.

IX. AXENFELD. A Further Observation of Corneal Curvature Opposite to that of Keratoconus (corrigible peripheric myopia and corrigible central hyperopia in the same eye). With six illustrations in the text.

X. Original articles from these ARCHIVES, vol. xxiii., Nos. 3 and 4, abstracted by R. Greeff.

Index of titles of articles mentioned in the report of progress for 1894 by authors' names.

VOL. XXIV. No. 4

ARCHIVES OF OPHTHALMOLOGY.

OBSERVATIONS ON CASES OF HEMICHROMATOPSIA, INDICATING THE NON-EXISTENCE OF A SEPARATE CORTICAL COLOR CENTRE.

By Dr. WARD A. HOLDEN.

THERE have been reported a considerable number of cases in which, with a normal field for large white test-objects, there was hemianopsia for colors. Most of the older writers agreed that there must be special separate cortical centres for the perception of form, light, and color, and that in the cases under consideration, the cortical color centre was exclusively involved. This supposition was defended particularly by Samelsohn, Landolt, and Wilbrand. Of late, however, there has been a tendency to depart from this teaching, and Ole Bull [1] and Otto Dahms [2] do not accept it, although their arguments against it are not altogether conclusive.

Some observations made a year ago convinced me that the supposition of there being a lesion of some special color centre in these cases was wrong. In some perimetric experiments, three patches, 1 *cm* square, one of pale green, one of saturated red, and one of saturated blue, were placed on separate gray cards, each card having the same light intensity as the color. The green could be seen 25° temporally in the horizontal meridian, the red 50°, and the blue 75°. Three gray patches were prepared on a white ground, having such an intensity that each gray patch had as large a field as one of the colors, and three black dots of different sizes on a white ground were so prepared that each dot was seen in a field as large as that of one of the colors.

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[1] *Perimetrie*, Friedrich Cohen, Bonn, 1895.

[2] *Ueber halbseitige Farbenblindheit*, Inaug. Dissert., Leipsic, 1895.

In a normal eye with a given illumination, the black dot, the gray patch, and the color were each seen over an equal area of the visual field, and this physiological relation was found also to hold good in most cases of amblyopia. Naturally it did not hold good in the cases of retinitis pigmentosa in which the color fields are reversed, and there was a slight discrepancy in some cases of optic-nerve disease. But in general, in pathological cases, blue will be seen as far in the periphery as the largest dot and darkest gray patch, red as far as the middle dot and patch, and green as far as the smallest dot and palest gray patch, and where the small dot and palest gray patch are not seen, no colors will be recognized, and this relation holds good also for the central and intermediate zones.

In two cases previously reported [1]—one with the field for white complete, and the other with a slight contraction to the temporal side, there was bi-temporal hemianopsia for all colors, and for all three black dots and gray patches; but with dots slightly larger and with patches slightly darker, the field in one case was complete and in the other nearly so.

In another case, with Jacksonian epilepsy and various cerebral disturbances, there was a partial homonymous hemianopsia of uncertain duration. For white test-objects, the line of demarcation between the blind and seeing halves of the retina in each eye ran in the vertical meridian in the lower half of the field and just below the point of fixation passed irregularly and obliquely to the periphery, leaving something over a quadrant of each retina blind. Tested with blue, the blind portion was slightly greater than when tested with white, and there was complete homonymous hemianopsia for green, and also for small dots and pale gray patches.

Thus it appears, in cases in which there is bi-temporal or incomplete homonymous hemianopsia for white, that the eyes are also unable to distinguish from white the black dots and gray patches which are not seen by the normal eye in parts of the field in which the corresponding colors are not recognized.

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[1] These ARCHIVES, Nos. 1 and 2, 1895.

A slight interference in conduction of any of the fibres of the visual tract, leads to an inability to recognize green or even red, or to distinguish slight differences in luminous intensity. A more marked disturbance in conduction leads to the inability to recognize blue, or to distinguish quite marked differences in luminous intensity. A greater interference with conduction prevents the distinguishing of white from black, and with complete interference with conduction even perception of light is lost.

Thus the recognition of color varies with the light sense, and the assumption of the involvement of a particular cortical color centre in cases of hemichromatopsia is not only unnecessary, but is palpably erroneous.

ON CHOROIDAL TUMORS.

By Dr. MITVALSKY, Prague, Austria.

Translated and abridged by Dr. Ward A. Holden.

(*With seven figures on Plates XVIII. and XIX. of Vol. XXVIII. Germ. Ed.*)

THE fully developed sarcoma of the choroid is usually a circumscribed true tumor projecting into the interior of the ball. There are cases, however, in which the tumor extends laterally, remaining flat and never projecting much above the surface of the choroid. Knapp and Brière in their treatises did not mention growths of this nature, and they were first described in Fuchs's monograph, where choroidal sarcomata were divided, in respect to form, into the circumscribed, having the ordinary tumor appearance, and the diffuse, in which a considerable portion of the choroid is slightly and uniformly thickened. Tumors of the latter form may finally fill the vitreous chamber, both by the continuation of the uniform thickening and by the development of processes of various size, whose apices finally meet, when this tumor cannot be distinguished from the circumscribed variety.

I would state, going further than Fuchs, that we may have flat sarcomata of the choroid that only develop laterally, and that may involve the entire uveal tract and yet never fill the interior of the eye, but may readily give rise to episcleral nodules.

Case I.—A flat sarcoma of the choroid, which in two years' growth attained the thickness of 1 *mm* and involved the outer portion of the choroid posteriorly.

N., aged sixty-six, consulted me January 3, 1892, stating that the sight of the right eye began to fail two years before without pain or inflammation. Three weeks before the visit the eye became inflamed and quite blind, and there was pain radiating into the forehead. The eye was glaucomatous, the interior cloudy, V = 0, and enucleation was done.

Microscopically (Fig. 1, Pl. XVIII.) the retina is seen to be detached and to extend forward in a single strand nearly to the lens, which is displaced forward, crowding the iris against the cornea. The sclera appears normal, but the choroid is somewhat thickened, particularly in the outer and posterior part, the transition to the normal choroid being very gradual.

Microscopically the diffuse thickening is seen to be due to the presence of a flat sarcoma composed for the most part of round, oval, and spindle cells, but having at some points the appearance of a dense fibroma and at others that of a soft fibroma, passing over into myxoma. Through a considerable extent of the tumor strands of new-formed unstriped muscle are found. The main portion of the sarcoma is composed of oval and spindle-formed cells not arranged in bundles (Fig. 2, *b*, Pl. XVIII.). The innermost layer lying on or near the chorio-capillaris consists of closely packed round sarcoma cells (Figs. 3, *b*, and 4, *b*, Pl. XVIII.), often arranged along small vessels, and probably transformed cells of the vessel walls. Considerable pigment is present (Fig. 2), but it is not in such quantity that the tumor could be called melanotic.

In the periphery of the tumor and also about the disc the tissue is purely sarcomatous. The part lying in the centre, which is thinner, has a layer purely fibrous (Fig. 3, *c*, Pl. XVIII.) and two layers purely sarcomatous (Fig. 3, *b* and *d*).

In many regions there are scattered oval and spindle formed cells with a granular intervening substance like a soft fibroma, or even a myxoma (Fig. 4, *c*).

There are also coarse bundles of unstriped muscle running meridionally in the midst of the sarcomatous mass. These are probably the result of proliferation of the muscle cells normally present just behind the ciliary body.

The iris and retina show degenerative changes.

We have here a remarkable case, and it is unfortunate that it was not seen early by some one competent to study it. The central portion of the tumor, the oldest portion, is a fibro-sarcoma, in some parts myxomatous, while most of the remainder of the tumor is purely sarcomatous. It is also noteworthy that this tumor had grown for two years, causing complete blindness but producing no external signs of irritation or inflammation. It is probable that the first inflammatory symptoms which appeared twenty days before his visit, were due to an acute outbreak of glaucoma.

CASE 2.—A second case of flat sarcoma of the choroid arising in the macular region and in the course of $3\frac{1}{2}$ years attaining only a thickness of 1.5 *mm*, leading to involvement of the retina and the formation of several secondary nodules in the anterior portions of the retina.

S. M., æt. forty-one, was seen January 26, 1889. Three months before the left eye had troubled him, and in the course of a few days central vision was lost. The eye was normal externally, —T $\frac{1}{2}$, media clear. The macular and paramacular region is occupied by a grayish yellow patch 6–8 disc diameters across and elevated 6 D in its most prominent portion. There was a central scotoma corresponding to the patch in form. As it was impossible to say whether the patch was a sarcoma or a plastic choroiditis with exudation, mercurial inunctions were employed and central vision returned in a measure.

March 2.—The macula could be seen and vision had increased to $\frac{6}{100}$, thus substantiating the diagnosis of central choroiditis.

In *December*, 1889, the eye became greatly inflamed, the central vision was lost, the choroid posteriorly was yellowish white, and there was perception of light only in the periphery. Later the vitreous was clear, and a flat yellowish opaque elevated patch occupied the macular region.

In *May*, 1892, there were constant headache and pain in and about the eye, + T $1\frac{1}{2}$, and enucleation was done.

In horizontal meridional sections the fundus is seen to be occupied by a thickening at some points as much as 1.5 *mm* thick, involving both choroid and retina, and having a smooth inner surface. Anteriorly in the region of the ora serrata there are small isolated nodules (Fig 5, Pl. XIX.). The retina which lies in position is thickened and opaque, and has many small elevations on its surface, which represent secondary disseminations of the tumor.

Microscopically the optic nerve is swollen and infiltrated, and the tissue of the disc is distinctly fibrous. This fibrous tissue extends from the region of the disc out over the fundus in a thick layer, giving a fascicular structure to the sarcoma. The choroid for a distance of 14 *mm* from the disc shows sarcomatous degeneration in its entire thickness, and this portion (Fig. 5, *a*, Pl. XIX.) is doubtless the oldest part of the sarcoma. Toward the equator the two nodules pass over into a cellular fibrous tissue containing groups and tracts of sarcoma cells.

The sarcoma masses in the retina show in their centres black tracts of pigment due to the proliferation of the pigment epithelium. The retina and choroid are in apposition as far forward as the ciliary body, and the sarcoma cells can nowhere be seen to have passed from the choroid into the retina, but the disseminated nodules anteriorly can be traced to a direct proliferation of the pigment epithelium.

The primary sarcoma nodules of the choroid have the appearance of a spindle-celled sarcoma of fascicular structure, passing over into a fibro-sarcoma.

It is probable that the propagation of the sarcoma was brought about by the preceding development of fibrous tissue, which then gave rise to the sarcoma cells.

The sarcomatous nodules of the retina, near the posterior pole, strongly resemble leiomyomatous tissue, and might be mistaken for it. The older nodules in the pigment epithelium have more the character of a fascicular spindle-celled sarcoma; the younger and smaller nodules show mostly small or large round cells.

The retina has undergone fibrous degeneration; the other parts of the eye are not markedly changed. It is remarkable in this case that the central scotoma discovered at the first visit disappeared under the use of mercury, so that the original change in the macula appeared to be rather a plastic inflammation of the choroid than a neoplasm. The absence of detachment of the retina is also noteworthy, although it is known that tumors of the macular region cause detachment less frequently than those of other parts.

Secondary invasion of the retina by choroidal sarcoma is rare, and may take place in two ways—either the portion of the retina covering the sarcoma is invaded directly, or a number of small sarcoma nodules form in the pigment epithelium, which proliferates and by undergoing a sarcomatous degeneration forms a direct communication between the sarcoma and the retina.

The genesis of the disseminated nodules in our case was evidently as follows: From the primary sarcoma at the posterior pole, the sarcomatous elements invaded the pigment epithelium and caused it to undergo a diffuse sarcomatous degeneration, then extending laterally caused dissemination nodules to form in the region of the ora serrata, and there was also secondary sarcomatous infection of the choroid anteriorly by the same process.

Explanation of the Figures.

Fig. 1.—Section of the eye in the first case, the sarcoma represented in black.

Fig. 2.—A portion of the periphery of the sarcoma: (*a*) chorio-capillaris, with remains of pigment epithelium, (*b*) sarcomatous tissue, (*c*) layer of unstriped muscle at the level of the supra-choroidea.

Fig. 3.—A fibro-sarcomatous portion from the macular region: (*a*) chorio-capillaris, (*b*) young sarcoma cells, (*c*) myxomatous tissue, (*d*) sarcoma tissue bordering the sclera.

Fig. 4.—A myxo-sarcomatous portion from the macular region, lettering as above.

Fig. 5.—Section of the ball in the second case, enlarged two diameters.

Fig. 6.—Anterior portion of a dissemination nodule of the sarcoma with broad tracts of proliferated pigment epithelium (*a*); the limitans choroideæ is pierced by the sarcoma tissue, which has then spread out in the choroid and supra-choroidea.

Fig. 7.—A portion of the macular nodule, in which the bundles of sarcoma cells are cut mostly in cross-sections, but also in part longitudinally.

Archiv für Augenheilkunde XXVIII.

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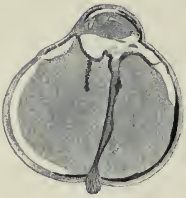

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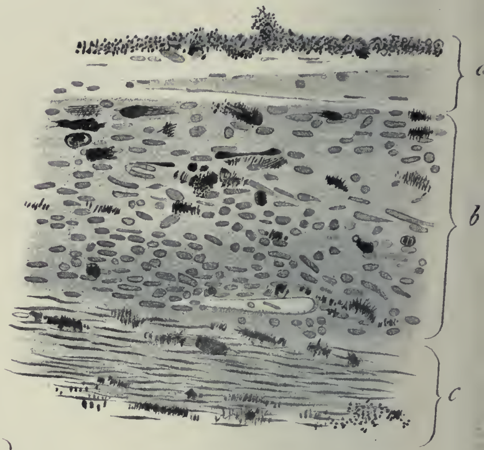

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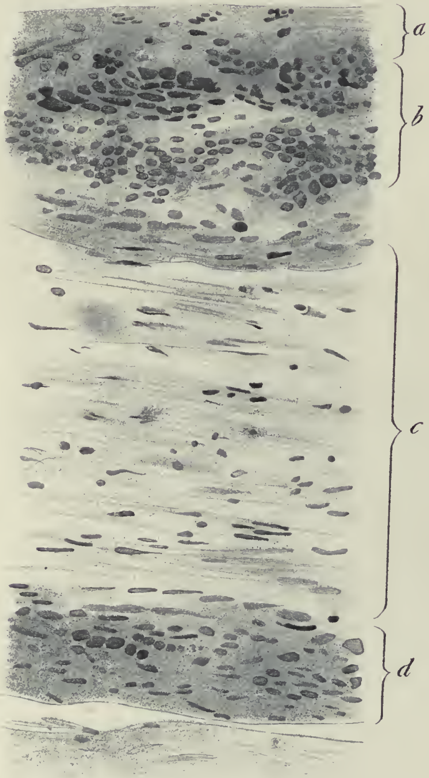

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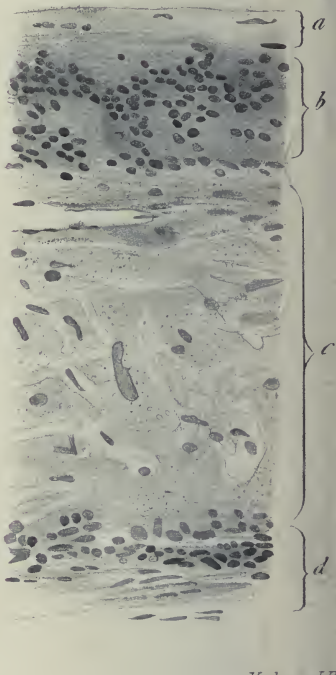

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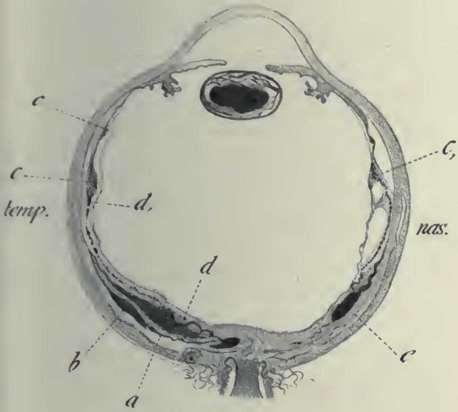

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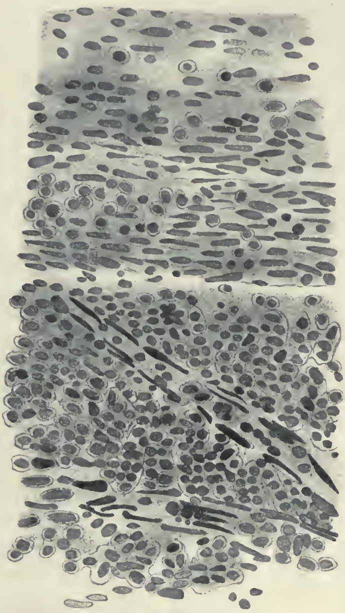

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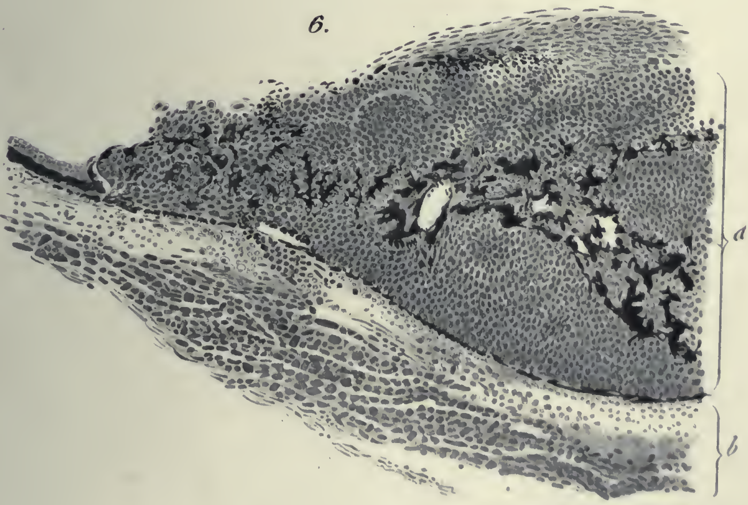

Wiesbaden

Lith. Anst. v. C. Kige, Göttingen

CONTRIBUTIONS TO THE KNOWLEDGE OF THE HISTOLOGICAL ALTERATIONS IN THE RETINA AFTER EXPERIMENTAL INJURIES.[1]

By Dr. A. TEPLJASCHIN, of Kasan, Russia.

Translated (Abridged) by Dr. Ward A. Holden.

(*With Figs. 1–28, Pl. XXI., XXII., and XXIII., Vol. XXVIII., Germ. Ed.*)

IN the last ten years our knowledge of the microscopic structure of the retina has been considerably increased. Recently established methods of microscopic investigation have made possible the study of the retina not only morphologically, but also with reference to the reciprocal relations of its elements.

As is well known, in the last connection, the most important results have been obtained in studying the retinal structure by the methods of Golgi and Ramon y Cajal, and that of Ehrlich. At the same time, most careful investigations have been directed to the retina in its embryonic development.

Our knowledge of the microscopic alterations in the pathological conditions of the retina remain, unfortunately, defective in spite of the fact that the knowledge of its normal structure and development has reached a high point. Experimental investigations on the microscopic changes in the retina after partial destruction, and on the regeneration of its elements under such circumstances, are of importance, and in this connection I refer to the work of Prof. Adamük in studying the histological changes in the retina of animals after the destruction of localized points, especially after wounds.

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[1] From the Histological Laboratory of the University of Kasan.

The possibility, even if remote, that, under the influence of trauma, changes may occur in those parts in relation with the retina, particularly the vitreous, must be considered, as well as the part played by the coats of the eye in the healing of perforating wounds of the posterior half of the organ.

Methods of Experiment and Investigation.

We have carried out our experiments on animals in the following manner: After pulling the eye either inwards or downwards with a pair of forceps, we plunge a discission-needle, five or six millimetres from the edge of the cornea, through the wall of the eye, avoiding injury to the lens. The needle is carefully run through the vitreous and by appropriate movements of the handle a cut is made in the retina.

In order to study the changes in the retina after wounds of that membrane, with simultaneous wounds of the choroid and the sclerotic, the same sort of penetrating wound is made as in operating in man for cysticercus or a foreign body in the vitreous, somewhat in the following manner: Having firmly fixed the eye we open the conjunctiva in a meridional direction, then pierce with a Beer's or Graefe's knife the wall of the organ about 4 *mm* from the margin of the cornea, and cut backward 4 or 5 *mm* in the same direction.

We have also, like other observers, experimented with cauterization of the retina. Seizing the conjunctiva close to the insertion of the superior rectus, we turn the eye forcibly downwards, and pierce with the incandescent cautery point the wall of the organ, at a point as far back as possible from the cornea, near the entrance of the optic nerve.

We have used rabbits for our experiments, and have employed the usual antiseptic precautions, both in the neighborhood of the eye and in the conjunctival sac.

No particular treatment was given to the eyes after the operation; they were examined from time to time with the ophthalmoscope, and then enucleated at periods of from 22 hours to 285 days in a total of about 50 experiments.

The eyes were removed from the animals whenever possible before the bodies were cold after chloroformization.

In case it were intended to make vertical sections, the eye and any sections already made were at once put into a fixing fluid, namely Flemming's osmic-chromic acid mixture, weak or strong, which enables us to study the nuclear division, the process of regeneration, and furthermore the fatty degeneration in the object studied. The specimens were, after remaining about 24 hours in this solution, hardened in alcohol, imbedded in celloidin, and cut with a microtome.

For staining the sections, particularly with a view to studying the nuclear changes, I used safranin, after the method which Bizzozzero has brought forward for the staining of karyokinetic figures. In addition to the study of vertical sections, for the sake of thoroughness we also studied the retina in the horizontal plane, and to accomplish this we have a very fine method of staining the nerve elements—the use of methylene blue. Ehrlich has introduced, for the purpose of staining the nerve elements, the method of injecting into the blood of living animals a solution of methylene blue, and such a method has occasionally been used for staining the retinal elements. Dogiel got very good results by staining the retina under the microscope with a $\frac{1}{16}$% methylene-blue solution. Good results can also be got if the method is modified as recommended by Arnstein for staining the nerves and nerve endings in the cornea. Our method was to fill the anterior chamber of the eye (cocainized) in a living animal with the solution of methylene blue, and after waiting one hour to enucleate it. We can get the same result by filling the anterior chamber of an already enucleated eye with the same solution, and keeping it, cornea upwards, in a constant temperature of 40° C. for an hour. In order to overcome some of the practical difficulties, I have made use of the following method: Taking a glass jar, half-full of hot water, and covering it with a piece of glass on which there rests a watch crystal, I am able in this to carry out the whole staining process with warm methylene-blue solution, as if on a warm stage. The eye comes immediately from the chloroformed animal to the warm fluid. The eye is further treated in the way described by Dogiel.

After the portion of the wall of the eye to be examined is placed carefully on a glass slide, retinal side up, five or six drops of a $\frac{1}{16}$ per cent. solution of methylene blue are dropped upon it, and it is protected with a cover-glass. From time to time it must be examined with a low power to note the progress of the stain, which is usually deep enough in most of the nerve elements in from fifteen to thirty minutes. As soon as the stain is deep enough, the excess of staining fluid is removed, and in many cases it is desirable to wash the preparation with normal sodium chloride solution by dropping a little on it. Then in order to fix the stain, the preparation still on the same glass is freely washed with a saturated watery solution of ammonium picrate. After twenty or more hours the fixing fluid is removed, and the preparation treated with equal parts of chemically pure glycerin and water, and a saturated solution of ammonium picrate. The retina must be protected from pressure by some bits of paper under the cover-glass. After one or two days the preparation is more or less transparent and ready for examination.

Histology.

Our task now consists in examining the changes in the retina seen in our specimens, but the close relationship between the retina and neighboring parts of the eye compels us to touch briefly upon some histological characteristics of those parts. Perforating wounds or cauterizations of the posterior hemisphere of the eye are always accompanied by a greater or less increase in the number of wandering cells of the vitreous, the increase being most marked four or five days after the operation, and the greatest number collect around the seat of the injury. The cells of the vitreous are, as is well known in the light of our present knowledge, amœboid bodies, wandering cells closely resembling the leucocytes that have left the blood-vessels. The question then arises whether these accumulated cells are only leucocytes, or, in part at least, new cells of the vitreous. The experiments of Pagenstecher have recently done much for the solution of this question. By the investigations of Bizzozzero, Ranvier, Klein, Stricker, Flemming, Lawdowsky, and others, the multiplication of leucocytes and wandering cells has been shown to belong to the type of ordinary cell-division. Peremeschko, Kultschizky, Marchand, Metschnikoff, Schpronk, and particularly Flemming, have shown from their observations that leucocytes multiply also by karyokinesis and karyomitosis. But since some observers have been unable to see the karyokinetic figures in the leucocytes, this method of multiplication for them has not been considered established.

In Fig. 1, Pl. XXI., are several leucocytes from a section made four days after the wounding of the eye; *a* and *b* are cells, the nuclei of which are dividing by karyomitosis, *c* is a cell which in addition to the karyokinetic figure is provided with a nucleus which is not dividing, *d* is a cell with a nucleus breaking up into fragments, *e* and *f* are cells with the signs of simple division, *g* is a cell with double round nuclei. We have in addition observed in the sections the picture of the leucocytes of the capillaries in the retina emigrating in the direction of the vitreous.

After the number of wandering cells in the vitreous had reached the highest point, about four or five days after the operation, there was a rapid diminution in the following days, and at the same time the evidences of the multiplication processes became less frequent. Even in the first days after the operation, regressive as well as progressive appearances were observed in the wandering cells. From the fourth or fifth day onward, a portion of the wandering cells of the vitreous assumed the characteristics of new connective-tissue cells which filled the perforating wound. Among these cells there were some showing karyokinetic figures. At a more or less late period after the operative interference there appeared not seldom on the inner surface of the retina (membrana limitans interna), in the region of the injury, a layer of cells of considerable extent. Viewed from above these cells appeared as flat, smooth endothelial cells possessing well marked oval nuclei. In flat preparations stained with methylene blue, one could see their formation from wandering cells. Frequently, late after the operation, a true connective-tissue layer was observed near the site of injury, where the retina already had undergone changes which in places was so tightly adherent to the retina that the radial supporting fibres of the retina passed over into this layer and helped form it. The development of connective tissue, after an injury to or a disease of the eye, occurs, as we know from pathology, not only in the periphery of the vitreous, but often in its deeper portions. Can this layer come from the wandering cells, or is connective tissue necessary to its formation? The investigations of Pagenstecher seem to show that connective tissue can be formed in the vitreous from wandering cells, and the same view is expressed in modern works on ophthalmology. Very recently, however, it began to be denied absolutely that leucocytes possessed the power to transform themselves into non-motile connective-tissue cells (Arnold, Marchand, Reinke, Nikiforow, and others). Our investigations seemed to show that the wandering cells of the vitreous might after traumatism of the eye become fixed connective-tissue cells.

Some days after the operation there could be seen proliferation of the endothelium of the capillary vessels. Later large flat cells with a nucleus and long processes appeared in immediate connection with the walls of capillary vessels or anastomosing with one another (Fig. 5, Pl. XXI.). These bore no similarity to the wandering cells of the periphery of the vitreous, and were, no doubt, vessel-building cells.

From the second day after the operation the retina became œdematous and thickened (Fig. 18, Pl. XXIII.), and the radial fibres swollen and thickened. This œdema gradually disappeared and was entirely gone in the course of a month or two. Shortly after the injury the retinal fibres showed large nodules which did not stain with safranin or with hematoxylin, and which were often grouped along the nerve fibre, forming varicose hypertrophies (Fig. 9, Pl. XXI.). The nature of these varicose hypertrophies is not yet fully understood. Under the influence of various reagents and after the death of nerve tissue the interfibrillar substance is deposited in irregular masses along the axis-cylinder, and these nodules are termed varicosities.

In methylene-blue preparations it is clear that after the retinal fibres are cut they undergo a complete atrophy both ascending and descending, the latter affecting also the ganglion cells. Some time after the operation, when the fibres were cut midway between disc and ora serrata, the nerve fibres were wanting in both directions (Fig. 13, Pl. XXI.).

In studying the changes after trauma it is interesting to find in how far the retinal elements of the adult animal preserve the power of regeneration. The changes in the ganglion cells were chiefly of a degenerative character, though karyokinetic figures were found (Fig. 14, Pl. XXII.) four or five days after the operation, but the figures were in the monaster stage. In the œdematous portions of the retina many wandering cells were found in the ganglion-cell layer (Fig. 17, *b*, Pl. XXII.). The changes occurring in the early stages in the inner reticular layer were chiefly due to the œdematous condition of the retina.

In the inner nuclear layer the nerve cells showed progressive and degenerative changes such as were found in the ganglion-cell layer, and karyokinetic changes were very frequent in the nuclei of the supporting fibres. In Fig. 15, Pl. XXII., the karyokinetic figure to the right is doubtless in the nucleus of a Müller's fibre, while the three others probably belong to nerve cells.

In the inner nuclear layer the retina is hydropic, and exudative matter is present in the pericellular spaces, and many of the cells have degenerated.

A few days after the injury subepithelial cells in a state of hyalin degeneration were frequently found near the ora serrata.

The outer reticular layer was simply œdematous.

In the layer of rods and cones these elements were soon destroyed, while in the outer nuclear layer there were frequent signs of progressive changes.

The confirmation of the belief that there are nuclei of the supporting fibres in the outer nuclear layer, explains the large karyokinetic figures that were frequently found here (Fig. 20, Pl. XXIII.). Thorough search failed to reveal such figures in the nuclei of the rods and cones in the outer nuclear layer, although there was an increase and a peculiar arrangement of the chromatin (Fig. 22, Pl. XXIII.). Other active changes appearing later, after the injury in the outer nuclear layers, seemed to show some power of regeneration in these cells and an attempt to reproduce the destroyed rod and cones (Fig. 23, Pl. XXIII.).

The cells of the pigment epithelium that were nearest the wound, mostly underwent a fatty degeneration (Fig. 26, *b*, Pl. XXIII.), while those farther away showed signs of proliferation by indirect nuclear division (Fig. 26, *a*, Pl. XXIII.).

The data obtained by our investigations show that after injury of the retina an inflammatory process begins in the region of the injury in which the retinal elements undergo changes of both a progressive and a regressive character. Changes of the latter character are found at the point of most severe inflammation both in the nervous and the visual elements. The progressive changes in the beginning are limited to regenerative processes, but a regeneration of nervous and visual elements that have been destroyed, does not occur. The supporting framework, however, proliferates, but this never leads to union of the margins of the wound. There takes place an atrophy of the retina and the choroid in the region of the retinal wound, due to a serous chorio-retinitis, after which the connective-tissue elements of the retina and choroid form a scar. In penetrating wounds the gap is closed mostly by proliferating epibulbar connective tissue, less by proliferation of choroidal tissue (Fig. 18, *b'*, Pl. XXIII.), and probably in small measure also by the wandering cells of the vitreous which become fixed connective-tissue cells.

Explanation of the Figures.

Fig. 1.—Perpendicular section through the vitreous 4 days after the operation, showing the wandering cells of the vitreous ;

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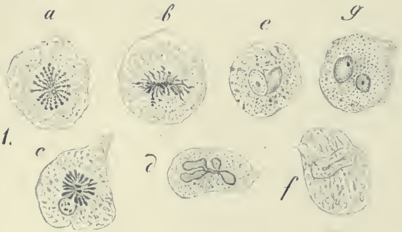

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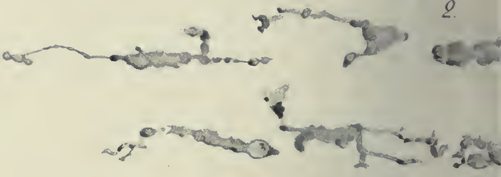

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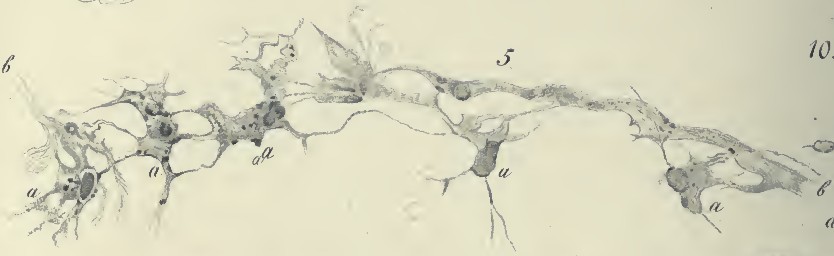

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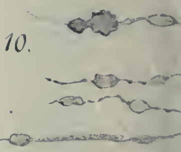

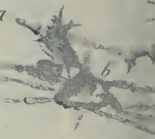

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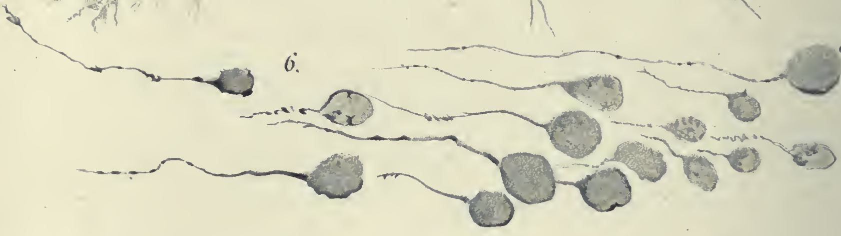

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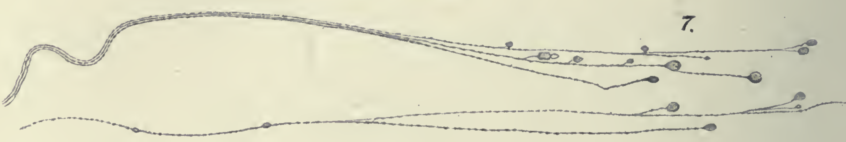

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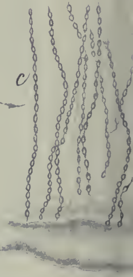

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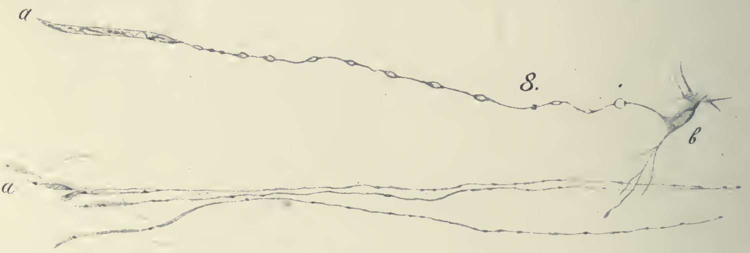

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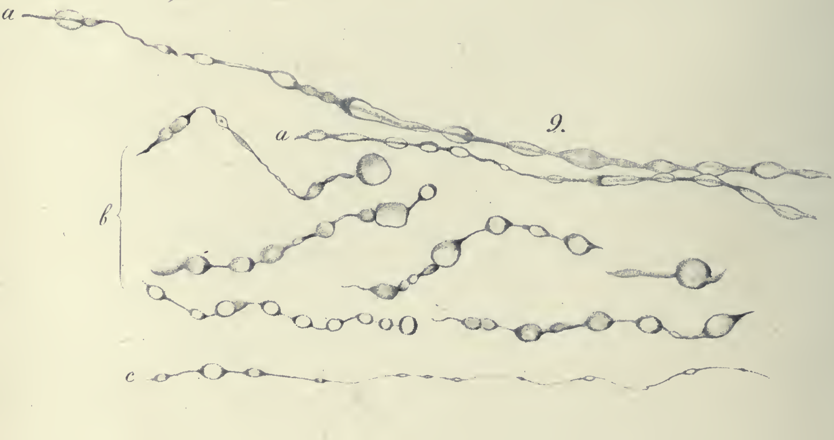

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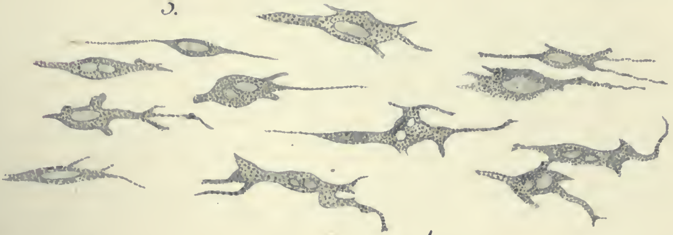

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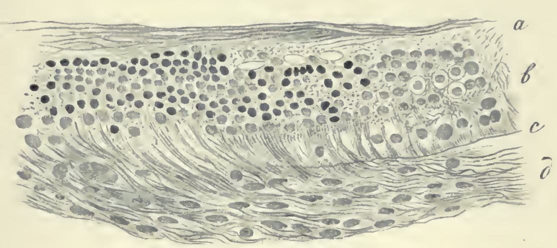

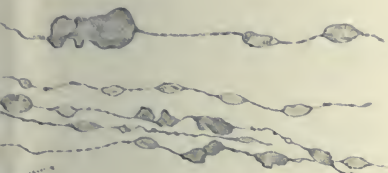

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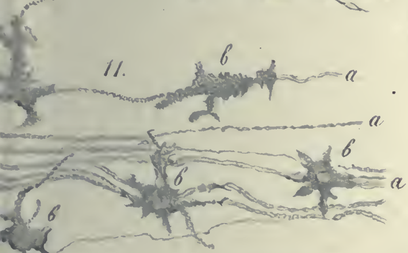

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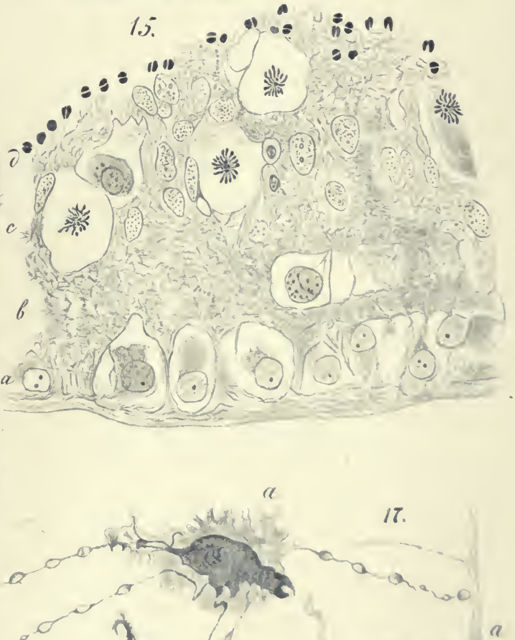

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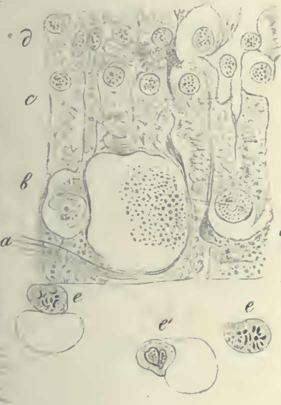

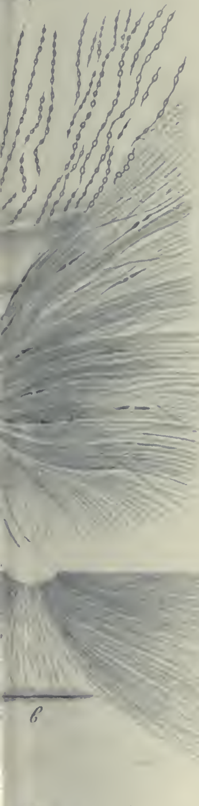

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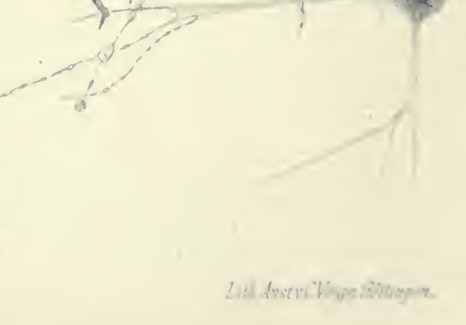

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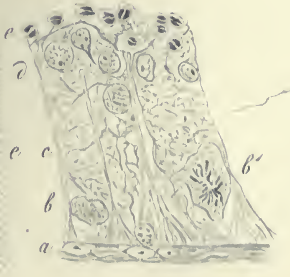

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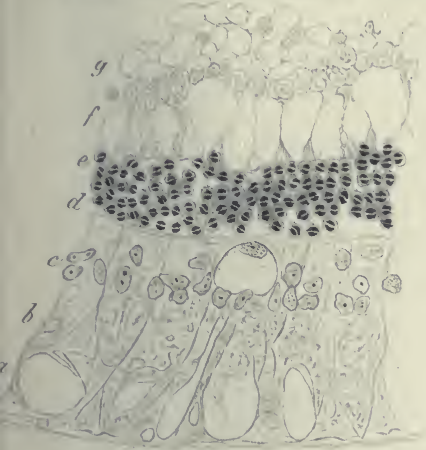
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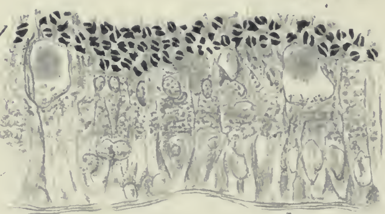
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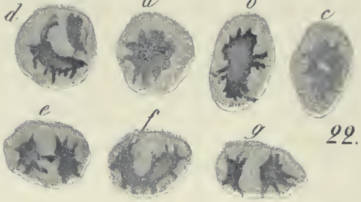
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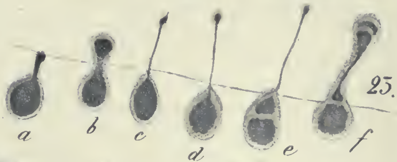
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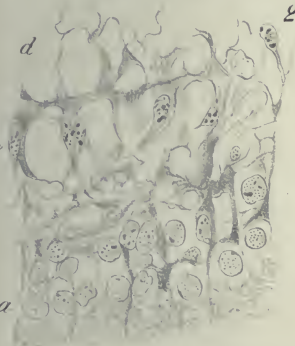
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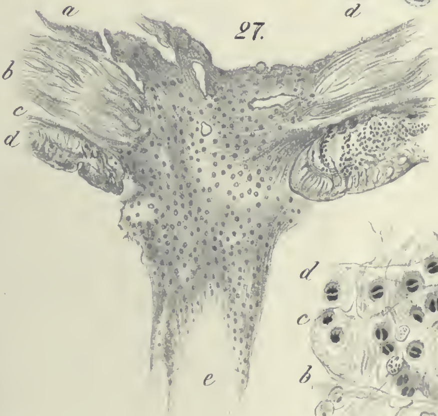
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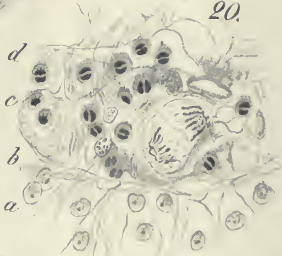
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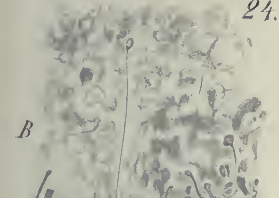
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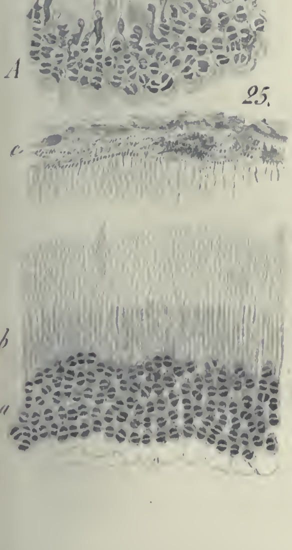
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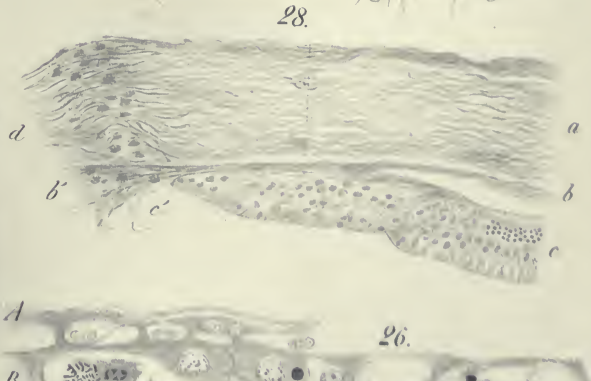
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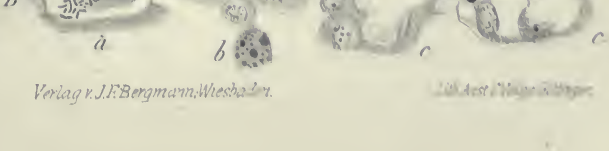
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a and *b* with mitotic changes, *c* with two resting nuclei, *d* with division of the nucleus by fragmentation, *e* and *f* with simple division of the nucleus, and *g* with two nuclei.

FIG. 2.—Vitreous 285 days after operation. The wandering cells of the vitreous have taken on the character of fixed cells.

FIG. 3.—A similar condition of vitreous cells in a human eye enucleated for cyclitis.

FIG. 4.—Vertical section of retina 89 days after operation. On the inner surface of the retina is a proliferation of connective-tissue in which Müller's fibres take part.

FIG. 5.—A flat preparation of a rabbit's retina 194 days after operation. Vessel-forming cells united to the walls of capillaries by processes or anastomosing with one another.

FIG. 6.—Retina of rabbit, flat, 8 days after operation. The central ends of cut nerve fibres with spherical terminations.

FIG. 7.—Rabbit's retina, flat, 4 days after operation. Finer fibres as in Fig. 6.

FIG. 8.—Rabbit's retina, flat, 4 days after operation. Peripheric portion of cut nerve fibres ending at *a*; *b*, a ganglion cell.

FIG. 9.—Rabbit's retina, flat, 194 days after operation, showing the peripheric sections of the nerve fibres after being cut near the disc. At *a*, slightly changed medullated fibres; *b*, nerve fibres with varicose hypertrophy.

FIG. 10.—Flat section of human retina with retino-cyclitis, showing varicose hypertrophy.

FIG. 11.—Flat section of retina 13 days after operation, showing the condition of fagocytosis in the degenerated nerve-fibre layer; *a*, degenerated nerve fibres; *b*, wandering cells.

FIG. 12.—Flat section of rabbit's retina 194 days after operation; *a* is a fold at the seat of the wound, *b* is an excavation in the disc, *c* the peripheric portion of nerve fibres with varicose hypertrophy.

FIG. 13.—Schematic sketch of the nerve-fibre layer of the retina 285 days after a wound parallel to the æquator. *a* is the disc; *b*, the scar; *c* is the triangular surface of the retina between disc and scar; *d* is the broad surface between the scar and the periphery of the retina, which is free from nerve fibres; *e* represents parts of the retina with a normal supply of fibres.

FIG. 14.—Vertical section of retina 4 days after operation. In the ganglion-cell layer *b* is a nerve cell in mitosis, *a* is the membrana limitans interna with wandering cells, *c* the inner reticular layer.

Fig. 15.—Vertical section of retina 4 days after operation, showing 4 karyokinetic figures, the one to the right being the nucleus of a Müller's fibre, the upper being the nuclei of subepithelial nerve cells, and the other two nearer the inner reticular layer. Some of the ganglion cells are degenerated.

Fig. 16.—Vertical section through the retina and vitreous 8 days after operation. In the ganglion-cell layer (*b*) is an exudation mass in the location of a ganglion cell; *e*, cells in the vitreous.

Fig. 17.—Flat section of retina 4 days after operation. *a'* is a ganglion cell surrounded by an exudation and with varicose processes; *a* is a normal ganglion cell; *b* are wandering cells.

Fig. 18.—Vertical section through the retina 5 days after operation, showing the hydropic condition of the retina.

Fig. 19.—Section of the retina 4 days after cauterization. In the inner nuclear layer are cavities containing subepithelial cells that have undergone hyaline degeneration.

Fig. 20.—Oblique section of the retina 4 days after operation, with one karyokinetic figure and irregular distribution of chromatin in other cells.

Fig. 21.—An oblique section of the retina 4 days after operation. On a level with the outer nuclear layer is supporting substance without visual cells, but with nuclei of supporting fibres.

Fig. 22.—An oblique section through the retina 4 days after operation. Various arrangements of chromatin in visual cells.

Fig. 23.—Vertical section of the retina 10 days after operation, showing nuclei of visual cells with processes passing beyond the mem. limit. ext. resembling the embryonic development of the rods and cones.

Fig. 24.—Vertical section through retina 15 days after operation, showing nuclei of the outer nuclear layer with extensions externally with thickened ends.

Fig. 25.—Vertical section of the retina of a black rabbit 90 days after operation, showing a distension of the rod and cone fibres.

Fig. 26.—An oblique section through the retina and choroid of a white rabbit 4 days after operation. *A* is the inner layers of the choroid, *B* the pigment epithelium; *a* is a pigment epithelial cell with a karyokinetic figure; *b* is an epithelial cell that has undergone fatty degeneration; *c* are cells with vacuoles.

Fig. 27.—A transverse section of a penetrating wound of the

ball 15 days after the operation. There is shown the proliferation of the young scar tissue which protrudes into the vitreous.

FIG. 28.—Vertical section of the walls of the eyeball at the margin of the scar 173 days after operation. *a* is the sclera passing into the scar, *d*, which fills the penetrating wound ; *b* is the choroid passing into cicatricial tissue, *b′* ; *c* is the retina, which gradually becomes thin and loses its visual and nervous elements ; *e* is the pointed margin of the retina, whose supporting fibres have taken nearly a horizontal direction.

A bibliography of 106 titles follows.

THE PALPEBRAL FORM OF SPRING-CATARRH.

By Dr. H. GRADLE, Chicago,

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IN the description of "spring-catarrh" of the conjunctiva by Saemisch in the treatise of Graefe and Saemisch (1874), in which this singular form of disease was first brought into general notice as a typical affection, no mention was made of any lesions of the palpebral conjunctiva. Subsequently Raymond[1] called attention to the co-existence of hypertrophic changes in the conjunctiva of the upper lid. In 1879, Horner again emphasized the importance of the conjunctival lesions in a dissertation by his pupil Vetsch.[2] In all but four of his thirty-seven cases the disease was accompanied by papillary hypertrophy of the tarsal part of the conjunctiva of the upper lid, and in some instances the changes on the lid surface were more pronounced than the circumcorneal lesion. In 1881 Swan M. Burnett[3] recorded his observations made in this country in which he also stated to have seen the conjunctival lesion in every case. Under the title "Periodic Disease of the Conjunctiva Allied to Hay-fever," I published[4] in 1886 the history of four cases in which a disease of the conjunctiva of the upper lid answering to the description by Raymond, Vetsch, and Burnett, recurred every warm season, *but without the characteristic circumcorneal lesion of spring-catarrh.* In this article I pointed out the periodicity of this disease and compared it with the course of hay-fever, like which it was promptly relieved by a trip to cooler regions during the summer.

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[1] *Annali di Ottalmologia*, Anno iv., quoted by Burnett.

[2] And in 1881 in his volume on eye diseases in *Gerhardt's Kinderkrankheiten.*

[3] Arch. of Ophth., vol. x., p. 414.

[4] *American Journal of the Med. Sciences*, April, 1886.

In 1888 Emmert[1] described three different types of spring-catarrh, viz.: (1) the form in which lesions are only found at the limbus; (2) the form in which the changes are limited to the conjunctiva of the lid; and (3) the mixed type. Amongst 29 instances of the disease seen by him, 8 belonged to the first type, 8 to the second, while 13 showed the changes in both localities. Since the publication by Emmert no special article has come to my notice, in which stress is laid on the occurrence of the palpebral form without disease at the limbus. Knus, in a Zurich dissertation 1889,[2] found the swelling at the limbus absent only 7 times in 64 cases collected from the records of the Zurich clinic, and in one of these the circumcorneal eruption made its appearance during the third year of the disease. He indeed expresses the opinion that the circumcorneal tumefaction will ensue in every case if the disease be observed long enough.

Of modern text-books, Nettleship (1890), Fuchs (1892), De Schweinitz (1892), and Juler (1893) recognize the occurrence of spring-catarrh with lesions limited to the conjunctiva of the upper lid. Noyes (1894) Norris and Oliver (1893), Berry (1893), and E. Meyer (1887) give satisfactory descriptions of the disease and enumerate the changes on the palpebral conjunctiva, but do not point out that the latter may occur without the circumcorneal eruption. De Wecker in his *Ocular Therapeutics* (Engl. translation, 1879) is rather vague as to the localization of the disease. Most of the other text-books in the English language omit all reference to spring-catarrh or give it but a very short notice.

In addition to the 4 cases previously reported I have seen 8 more instances of this disease. Of these 12, one had a very slight circumcorneal tumefaction consisting of three discrete nodules, not larger than small phlyctenulæ. The other 11 patients showed no lesions except on the tarsal portion of the conjunctiva of the upper lid. Several of these cases had been diagnosed as trachoma by other oculists. The liability to this mistake and the comparative silence on this subject in American literature seem to me a sufficient reason for again referring to it.

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[1] *Centralb. f. prakt. Augenheilk.*, 1888, p. 71.

[2] Abstr. in *Centralb. f. prakt. Augenheilk.*, 1891, p. 267.

The complaints of the patients are comparatively severe. They begin with the onset of warm weather and last until the end of September or October. One young lady only, with very marked hypertrophy existing two summer seasons, was not entirely free from annoyance last winter. The discomfort increases with the temperature. A few cool days give relief. The annoyance consists in dread of light, itching, burning, and smarting pain, mostly with watering of the eyes, usually worse in the afternoon. Reading is interfeared with in proportion to the discomfort experienced. All my patients had secretion in the form of stringy mucus or pus. The disease affects always both eyes, although there may be some differences in the intensity on the two sides.

The appearance of the conjunctiva varies considerably with the length of time the disease has lasted. In two instances the conjunctival surface was smooth and but slightly injected when the trouble began. There was nothing characteristic to be seen the first summer, but towards fall the surface became slightly velvety in one of the patients. The next year the conjunctiva of the first patient presented the appearance of slight follicular conjunctivitis, while the second one had by this time marked papillary hypertrophy. In both patients the lesions became more pronounced the following season. Five other patients who were seen during the second season of their complaint, showed changes varying between a fairly smooth but reddened surface with underlying minute follicles to decided roughness due to papillary proliferation. The third or fourth year the hypertrophy in the form of papillæ, sometimes distinctly pedunculated, reaches its full extent. Two patients seen during the thirteenth and twentieth yearly attack had the same lesions that I had seen in others in the third or fourth year. From the second year the surface usually presents an opaque appearance, as most of the writers describe it, as if covered with a thin layer of milk. But this description does not apply in every instance. In the later years I have seen repeatedly the surface of a pale yellowish hue with very slight vascularity.

The upper fornix of the conjunctiva is either normal or shows but very slight papillary hypertrophy. The surface of the lower lid may be somewhat reddened but has never been involved in the disease in my patients. With the exception of one young man with very slight circumcorneal hypertrophy, all my patients showed a normal ocular conjunctiva. European observers have spoken of a circular streak of corneal opacity like a (premature) arcus senilis as a frequent occurrence in spring-catarrh. This I have never seen.

According to microscopical researches by Emmert, Knus, and Fuchs the papillary excrescences consist of proliferated connective tissue covered with multiple, thickened layers of epithelium. Fuchs speaks of hyaloid degeneration of both connective tissue and blood-vessels. The circumcorneal tumefaction of spring-catarrh consists, according to all observers, of epithelial proliferation growing into the connective-tissue basis in the form of plugs resembling the structure of cancroid. The marked growth of the epithelial cells makes it probable that the primary starting-point of the disease is in the epithelium itself, at least as far as the circumcorneal lesions are concerned. But in the case of the palpebral conjunctiva the microscopic evidence is not so decisive. Both Knus and Fuchs state that they have searched in vain for micro-organisms.

During the cold season the patients' complaints cease entirely (with the exception of one girl previously mentioned). In one patient seen at intervals during the first four years, I found the conjunctiva perfectly normal in winter. In the girl repeatedly referred to, the velvety conjunctiva became smooth and normal the first winter, but after marked papillary hypertrophy had occurred the appearance did not change much the second winter. Hansen Grut[1] and Fuchs state that they have seen the conjunctival lesions become smaller, but not recede entirely during winter. The circumcorneal tumefaction is generally said to disappear nearly or quite entirely during the cool season.

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[1] *Nordsk ophth. tidskrift*, 1888, abstr. in *Centralbl. f. prakt. Augenheilk.* 1888, p. 380.

No instances have as yet been observed in which the palpebral lesion was followed by any complications. But, nevertheless, the disease is a serious matter to the patient on account of its indefinite duration. However, Emmert, H. Grut, and Fuchs speak of its ceasing entirely in some patients after a number of years, sometimes without known reason, sometimes in consequence of a change of residence. But the number of such recoveries is very small compared with the number of patients recorded.

While spring-catarrh in one or the other form has been seen in every civilized country, there are evidently regions comparatively immune against this disease. Emmert calls attention to this fact in speaking of its geographical distribution in so small a country as Switzerland. As far as I can learn I was the first to state (in 1886) that the annual attack can be stopped temporarily or even permanently by a trip to a cooler locality, especially to spots known to be free from hay-fever. Four of my patients have found relief at the sea-shore, at Mackinaw, in the mountains of Colorado, and in Northern Minnesota (Lake Minnetonka). It is a peculiarity worth noting that in the West where, according to the experience of other colleagues, spring-catarrh is not a common disease, eleven of twelve patients suffering from this affection presented only the palpebral lesions and no involvement of the limbus region.

In none of my patients could any clue be found throwing any light on the etiology of the disease. The disease begins early in life, mostly during childhood. Four of the twelve spoke of one or two attacks in previous years of irritation of the respiratory passages during the summer, which had suggested hay-fever. But not one of them presented the history of annually recurring typical hay-fever.

No writer has as yet recorded any curative treatment. Abscission of the larger papillæ or their destruction by means of a pointed burner is recommended in extreme cases, but no one has yet recorded any cure by this method. In two instances I have obtained decided temporary relief by an energetic use of Knapp's trachoma-forceps, but after all it was but a temporary gain. In one of the same patients and in two others I have given relief by a few repetitions of friction of the diseased conjunctiva with a hard cotton swab moistened with corrosive sublimate solution of 1 in 100 (Keiming's method). But as far as my experience goes no treatment will do more than palliate so long as the high temperature persists, while when the season or at least the temperature declines, any of the applications customary in follicular conjunctivitis seem to hasten the recovery, so that the patient really gains in time by submitting to treatment. Nitrate of silver (2 %) and the frequent and free use of boric acid solution (4 %) mitigate the suffering as long as these applications are continued. Fuchs endorses as a palliative measure a collyrium of one drop of acetic acid in 20 grammes water, as recommended by Van Millingen of Constantinople. My own experience with this solution has not been extensive enough to be conclusive.

A CASE OF DIPHTHERITIC CONJUNCTIVITIS PRESENTING UNUSUAL COMPLICATIONS, TREATED BY ANTITOXIN.

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ALTHOUGH we all know what is meant by diphtheria of the larynx and pharynx, nevertheless the case is quite different with diphtheria of the conjunctiva. Even to-day there is much confusion as regards the nomenclature of the disease. What one author calls diphtheritic conjunctivitis and considers as a special disease,—sui generis,—regarding the formation of pseudomembranes only as *one* important symptom among others, others term the same disease pseudomembranous conjunctivitis, attaching the most importance to the formation of pseudomembranes. Pseudomembranous conjunctivitis includes not only the diphtheritic, but also, as soon as pseudomembranes are formed, the blennorrhoic, the trachomatous, and acute conjunctivitis.

Some authors, like Knapp, make a sharp distinction between croupous and diphtheritic conjunctivitis ; others, like Uhthoff, consider both as the same disease, differing only in degree, since both diseases are caused by the same morbific agent, the diphtheritic bacillus.

Until the middle of this century diphtheritic conjunctivitis was entirely unknown. A. von Graefe,[1] was the first, who from observations made on forty cases of diphtheritic conjunctivitis differentiated this disease from the catarrhal and blennorrhoic conjunctivitis, notwithstanding the fact that in all three diseases pseudomembranous exudations occur. He was the first to present the *clinical* picture of this disease.

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[1] *Arch. f. Oph.*, Bd. i.

The *pathological anatomy and symptomatology* of diphtheritic conjunctivitis and its kindred form, the croupous conjunctivitis, are described in Graefe-Saemisch's *Handbuch* and their clinical picture was drawn later in a classical way by H. Knapp.[1]

The *etiology* of this disease was unknown till 1883, when Klebs,[2] and in the following year Loeffler,[3] described and isolated a characteristic bacillus found in the throat of patients who had died from diphtheria. In 1886 and 1889, the same bacillus was found and isolated in diphtheritic conjunctivitis by Babes[4] and Kolisko and Paltauf.[5] Recently, in 1892, Uhthoff[6] was the first, who in his description of cases of croupous conjunctivitis showed, that the Klebs-Loeffler bacillus was etiologically responsible for the affection.

Attention to *the therapy* of this disease was called by the recent discovery of antitoxin by Behring. But while the literature upon diphtheria of the throat became flooded with publications for and against the value of antitoxin, the number of publications referring to the treatment of diphtheritic conjunctivitis by antitoxin is comparatively small. To my knowledge there are but few authors, viz.: Jessop, Santos, Coppez, Morax, McGregor, and possibly one or two others, who collectively published about fifteen cases. I consider it therefore not superfluous to describe another case. I do this because, as far as I am aware, this is the first case of diphtheritic conjunctivitis treated by antitoxin published in the United States, and also because there were some accompanying complications, which I did not find in any of the cases previously published.

F. G., thirteen months old, developed measles on June 3, 1895, which ran a normal course. Five days later, June 8th, the left eye became swollen. About thirty-six hours after this the patient was brought to the office of Dr. Knapp, who applied a protective bandage over the unaffected eye and placed some cotton in the ear. He then kindly transferred the case to me and advised me to make iced applications to the lids, instil boric acid solution, and employ hypodermic injection of antitoxin. A few hours later I saw the child at its home and found the following condition :

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[1] ARCH. OF OPH., Bd. xii.
[2] *Verhandlung des 2d Congresses f. in. Medicin.*
[3] *Mittheilungen aus den Kaiserl. Gesundheitsamtes*, Bd. ii., 1884.
[4] *Progrès médical*, 1886, 1888.
[5] *Wiener klin. Wochenschrift*, 1889, No. 8.
[6] *Berlin. klin. Wochenschrift*, 1894, No. 34.

A well-nourished child, still showing signs of measles in the shape of pale patches over the entire body. Temperature, 105°. Pulse, 120. Respiration, 35. The pharynx slightly reddened, but clean. The right eye protected by an occlusive bandage. The upper lid of the left eye immensely swollen, red, hot, and of wooden hardness, overlapping the lower lid about one-quarter of an inch. On the cutaneous surface of the lower lid, about half an inch distant from the margin there was an ulcer with diphtheritic margins and filled with a whitish diphtheritic mass. The ulcer was about one and a half inches long, a quarter of an inch wide, a quarter of an inch deep. Questioning the parents about this ulcer, I was told that another physician who had previously treated the child, had forcibly opened the eye. They therefore thought that he had probably made the wound, which later developed into this ulcer. All around the ulcer on the lower lid, and farther down on the cheek, there were eczematous excoriations. The conjunctiva of the upper and lower lids and of the eyeball appeared covered with whitish, shining, fatty pseudomembranes, burrowing deeply into the conjunctiva, so that they could not easily be detached. The cornea was surrounded and partly overlapped by the chemosed conjunctiva. The surface of the cornea looked hazy and smoky. On account of the stiffness of the lids it took considerable force to open the eye in order to see the cornea. The gland behind the ear was swollen. I injected 5 *cc* of Behring's antitoxin No. 11, instilled some boric acid solution, and ordered iced applications every two minutes.

The next day, *June 11th.*—The child vomits frequently. The nose becomes filled with diphtheritic masses. Temperature and pulse remain unchanged.

June 12th.—The surface of the cheek below the ulcer, which I have mentioned above, becomes red, hot, and swollen. The swelling being limited above by the ulcer of the lower lid and by the malar bone, on the right by the sulcus naso-labialis, below by a line drawn from the aperture naris to the lobulus auris, and on the left by the ear. Small diphtheritic patches appear on the tonsils. The general condition and the condition of the eye remain unchanged.

June 13th.—The morbid change in the throat has progressed, both tonsils and the uvula are covered with thick diphtheritic membranes.

June 14th.—The diphtheritic masses on the conjunctiva of the eye, on the ulcer of the lower lid and in the cheek begin to dissolve. I cleanse these wounds every two to three hours by irrigation with boric acid solution. The cornea is clearing up, but an abscess is formed in the lower part. The condition of the throat is still bad.

June 15th.—The discharge of pus is more abundant, and the blennorrhoic stage of the diphtheritic conjunctivitis begins. In the cornea a deep ulcer takes the place of the abscess. As the upper lid becomes cyanotic I change the iced compresses to Priesnitz applications. The general condition, the temperature, and the appearance of the throat remaining unchanged, I again inject 10 *cc* of Behring's antitoxin No. 11. On the same day an inspector of the Board of Health takes cultures from the conjunctiva, the ulcer, and the throat. Two days later he notifies me that true diphtheria bacilli had been found in all three cultures.

June 16th.—The upper lid becomes necrotic over a small area, through which pus escapes. The ulcers of both lids are now discharging an immense amount of pus. The pus of the extensive cellulitis of the left cheek is pressed out several times a day through the ulcer of the lower lid, irrigated and tamponed with antiseptic gauze.

June 17th.—The abscess of the cornea having burst, the iris lies in the wound. The throat begins to clear. The discharge of pus from the cellulitis of the cheek and from the conjunctiva of the lids is still very abundant.

June 18th.—The immense discharge of pus from the cheek and lids requiring constant cleaning, I consider it advisable to transfer the child to the Riverside Hospital. The rest of the history is given to me by Dr. Steinzik, house physician of the hospital, to whom I hereby express my obligations. The discharge of pus from the cellulitis of the cheek did not cease, so that Dr. Steinzik was compelled on June 22d to make a counter-opening near the ear. In spite of this, on June 24th the cellulitis spread to the forehead and from there to the other cheek. Now lead water applications were made, which arrested the progress of the cellulitis. The temperature varied constantly from 102° to 105°. On June 29th the child died of general prostration and weakness of the heart.

Summing up the observations made on this case, we find that the most interesting feature undoubtedly was the accompanying cellulitis of the cheek, which complication I could not find in any of the published cases of diphtheritic conjunctivitis. The origin of the cellulitis was probably this. Eczema faciei is often found accompanying diphtheritic conjunctivitis. We find it in the cases published by Graefe, Hirschberg, Uhthoff. Horner[1] says: "Eczema faciei is especially important, because it is very often the origin of diphtheritic conjunctivitis." How carefully the eczematous eruptions must be watched, treated, and protected from every injury, is illustrated in the above case. The lower lid was surrounded by small eczematous excoriations, the heavy upper lid, which was covered with diphtheritic pseudomembranes, overlapped the lower lid and macerated its outer eczematous surface. The forced opening of the lids by an unskilled physician, who wanted to look at the cornea, caused the lower lid to be slightly injured. This wound became infected by the overlapping of the diphtheritic conjunctiva of the upper lid, and in this way caused the immense cellulitis, which affected nearly one half of the left cheek and afterward spread to the right side.

I found in all cases of diphtheritic conjunctivitis with corneal complications, reported by Uhthoff, Schirmer, Graefe, and others, that in the beginning the whole cornea was hazy, and afterward an abscess began to form in its *lower* part. The abscess in the cornea was similarly formed in my case. It therefore seems to me, that the pus cells of the inflamed cornea, by reason of their gravity, accumulate in the lower part of the cornea, thus producing the abscess.

Although it is a generally accepted fact, that the application of ice in the treatment of diphtheritic conjunctivitis is very important, nevertheless this case shows that caution in those applications is necessary. The lids in diphtheritic conjunctivitis are intensely infiltrated with the exudative masses. The blood-vessels are compressed, hence the white, shining appearance of the conjunctiva. There is a stasis in the circulation. By continuous ice applications the small vessels in the outer skin become more contracted. The circulation is seriously interfered with, and necrosis of the skin is the consequence, as was seen in my case.

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[1] Aertzl. Centr. Verein in der Schweiz, 1874.

Concerning the serum treatment, I would state that, at least in the case under observation, it did not fulfil all the promises and sanguine expectations of Behring and his followers. The value of the serum as a preventive remedy was not verified. We have seen that, notwithstanding the serum was injected thirty-six or forty-eight hours after the disease was first noticed, the diphtheria inflammation progressed unhindered from the conjunctiva, probably by the canalis naso-lachrymalis into the nose, and later through the cavitas naso-pharyngea into the pharynx. This mode of spreading from the conjunctiva into the nose and throat we find also in cases mentioned by Vennemann, Despagnet, and Gerke.

It is true that the second, the blennorrhoic, stage of the diphtheritic conjunctivitis had already begun on the fifth day, and that the condition of the pharynx had also improved after the seventh day, but the question arises, whether the antitoxin was responsible for the improvement or not. It did not impress me, that the 20 *cc.* of Behring's antitoxin used in this case influenced in any way the course of the disease. W. H. Jessop[1] has also noticed a relapse after the use of antitoxin. He says: "Curiously enough, there was an apparent relapse in the first case, accompanied by the formation of a fresh patch of membrane on the left tonsil." Beside Jessop there were other authors, as Kossel, Wolf, Goebel, who reported relapses after the use of antitoxin.

Although no conclusions as to the value of a remedy can be drawn from a single case, nevertheless the case reported above will at least strengthen the opinion that we can not rely upon the antitoxin as a sure remedy.

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[1] *Medical Press*, 1895.

Excluding the antitoxin, the most important points of the treatment in diphtheritic conjunctivitis are: careful iced applications in the first stage, warm applications in the blennorrhoic stage, cleaning of the eye, irrigations, and instillations of weak antiseptic solutions.

A DERMOID TUMOR INVOLVING THE CORNEA, WITH REMARKS.

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GROWTHS involving the cornea and its immediate vicinity require removal because of the deformity they occasion and to preserve the eyesight. In the present patient the deformity was so noticeable that it became a great source of annoyance, through the inmates of the institution, where she holds the position of nurse, frequently jeering and poking fun at her under slight provocations of anger.

History.—It was this sensitiveness that led Allie E., twenty-six years old and unmarried, and a nurse in the Southern Indiana Insane Asylum, to consult me June 20, 1894, because of a tumor growing from the cornea of the right eye. Since birth she states the trouble dates, and has gradually grown from the corneoscleral junction, temporal side, more and more into clear cornea until it now reaches half-way to the pupil at ordinary dilatation. A dense, sclerosed grayish-white band demarcates clear corneal tissue from tumor. Outward beyond the corneoscleral junction the growth extends some 5 *mm*, and involves the sclera. The longest measurement of the tumor is from 10 to 12 *mm*, and its greatest width is from 8 to 9 *mm*. In color it is reddish-gray, has an uneven surface, is firm to the touch, not very sensitive, and so intimately connected with the cornea that it is only a very little displaceable. Projecting from its centre are two stiff, dark-colored hairs, resembling eyelashes.

Under cocaine and in the presence of Drs. Snyder and Rypens, an *operation* was undertaken for its removal. With a straight keratome the tumor was first dissected from a very close and intimate connection with the underlying cornea, and then in a similar way detached from the sclera, leaving the latter, at the site of removal, very thin, bluish-white in color, and semi-transparent. The hemorrhage nearest the clear cornea was trifling, but increased more and more as the limbus was reached. The scleral conjunctiva was united with a single suture, and the sclerosed band separating the transparent cornea from that occupied by the growth was touched throughout its length with the galvano-cautery. The healing was gradual but uninterrupted, with much clearing of the dense corneal opacity.

The report on the *microscopical appearance of the tumor*, made and sent to me by my friend, Professor Linsley, of the University of Vermont, is that it is of epithelial origin, of slow growth, and non-malignant. It is not typical of any class of tumors, but does resemble a dermoid cyst. There were a few hair bulbs in the middle of the sections, and pavement-epithelial cells were found beneath the Malpighian layer of the conjunctiva. It is fairly well supplied with blood-vessels. It is not a papilloma.

Development.—Almost without exception it is agreed that the new growths met with in this region of the eyeball have their origin primarily in the conjunctival limbus, and not in the cornea proper. The explanation for this is found in the peculiar method of blood supply, which is derived from loops of vessels which penetrate only the corneal periphery. About the only congenital tumor found growing from the cornea or its immediate vicinity is the teratoma or dermoid cyst. Its etiology, of course, is the same as that when it occurs in other parts of the body, namely an inversion of the epiblast during embryonic existence and an islet deposit of cells which should have made skin tissue, hair follicles, and sebaceous glands in another part of the body. It is true Van Duyse's explanation is that the amnion was at one time united with the surface of the eyeball, and eventually through constriction and separation there remained behind at the original point of contact amniotic cells, from which later the dermoid cyst develops. This theory, however, is not generally accepted.

The *histological construction* of a dermoid tumor, as the name indicates, is that of skin tissue, the superficial layer being frequently horny and dry and of uneven thickness. Acinous and sebaceous glands are met with in the deeper structures, together with hair follicles. The hair, as a rule is very fine, with little pigment. In the present instance, however, it was deeply pigmented, and resembled very much the eyelashes. Fibrous connective tissue with underneath a cushion of fat makes up the tissue next to the cornea. Indeed, fatty tissue not infrequently predominates, so that the growth is more properly a lipoma rather than a dermoid. Nerves and blood-vessels are sparsely found, and are most plentiful in the scleral portions of the growth. The growth of dermoid tumors is, as a rule, very slow, and their removal is oftenest called for because of their unsightliness.

The *operation*, at times easy, is again very difficult, and requires much care that the anterior chamber of the eye be not opened. A too superficial excision may be followed by recurrence and a more rapid and larger growth than the original tumor.

In association, not infrequently one finds *other anomalies* in development, such as coloboma of the eyelids or a marked degree of amblyopia. Our patient presented this latter associated with mixed astigmatism. Vision in the eye being $\frac{15}{70}$, improved slightly with — 1.5 cyl. ax. 180, and likewise with + 0.5 cyl. ax. 90. Besides, the patient has a lack of development of the entire right side of the face, the last molar tooth in the upper jaw never having developed. The skin over the right malar bone and extending down over the lower maxilla is nodulated and very much deformed.

August 2, 1895, patient by request presented herself for examination. There has been no return of the disease and the cornea is cleared up wonderfully, so that the iris is distinctly seen through the former seat of the growth; the dense line of demarcation in the sound cornea still remains.

A CASE OF SUCCESSFUL EXTRACTION OF A PIECE OF STEEL FROM AN IRIS IN WHICH A PLASTIC INFLAMMATION HAD BEEN ESTABLISHED, WITH PROMPT SUBSIDENCE OF THE INFLAMMATORY REACTION AND RESTORATION OF FULL VISION.[1]

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On April 18, 1895, I was consulted by Chas. C——, aged eighteen, a baker by trade, about his left eye. He gave the following history: While opening a barrel eight days previous he was struck in the left eye by a bit of something, which produced quite an acute pain at the time. The pain, however, subsided in a short time, consequently he resumed his work without consulting any one. In the course of a few days the pain returned, the eye became red, and he suffered somewhat from photophobia. He then consulted an oculist, who instilled some atropia sulph. solution and told him to let the eye alone.

On the eighth day after the accident when I saw him the cornea was clear, and a careful inspection failed to show any trace of trauma save a very slight hazy spot in the upper-outer quadrant near the pupil. There was some congestion of the ocular conjunctiva, considerable ciliary injection, and the pupil was dilated, due to the use of atropia. A careful examination of the iris under oblique illumination revealed a minute black object in the upper-outer quadrant surrounded by plastic material, the rest of the iris was somewhat thickened and inflamed.

The patient was told of this and impressed with the seriousness of the case and advised to have the foreign body removed, but when told that an operation would be necessary, he flatly refused.

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[1] Read before the Pittsburg Academy of Medicine, September 16, 1895.

He returned, however, two days later suffering more pain and intense photophobia.

An inspection of the eye at this time showed a decided deposit of lymph or perhaps pus in the anterior chamber—in other words, there was a decided hypopion. At this time he consented to an operation. I sent him to the hospital at once, and operated the same afternoon, under cocaine anæsthesia, assisted by the house surgeon. When I began I had in mind to enter the anterior chamber with a keratome from the outer canthus, a little below the foreign body, and then to grasp it with an iris forceps and remove it. The cut with the keratome was done nicely, but when I attempted to remove the foreign body I failed to dislodge it from the iris. After several unsuccessful attempts I caught up the foreign body and some of the iris, withdrew it and cut it off; in other words, I made a free iridectomy, including the foreign body. The patient was immediately removed to his bed and hot boric acid applications ordered. A grave prognosis was given. On the second day after the operation I was more than gratified to find the anterior chamber fully restored, the corneal cut firmly healed, the cornea clear, and that the hypopion had disappeared entirely. The iris resumed its normal appearance and the ciliary injection passed away.

The patient left the hospital on the sixth day (sixteenth since accident) with the eye doing nicely and vision almost fully restored. A few weeks later the vision equalled that of its fellow eye, and has continued so up to the present writing.

The case is interesting and instructive; interesting, on account of the good result after a plastic, and in fact almost purulent, form of iritis; instructive, because it shows what prompt operative interference and careful after-treatment may do.

It is also instructive because it has taught me that in a similar case I would enter the anterior chamber with a keratome immediately over the area of the foreign body and not above or below it, because a failure to dislodge the foreign body would require a smaller iridectomy for its removal than otherwise.

REPORT AND EXPERIENCES OF AN ITINERANT CATARACT OPERATOR AT THE END OF THE NINETEENTH CENTURY.

By Dr. O. WALTER, Odessa, Russia.

Abridged and translated by H. Knapp.

WHEREAS the larger towns of Russia are well supplied with oculists, the number of eye specialists in the whole vast empire of 112,000,000 is, according to Belljarminow, only 139, to which 283 physicians may be added, who also practise ophthalmology. Districts of two or three millions of inhabitants are without a single ophthalmic surgeon, whereas in the town of Odessa, with 350,000 inhabitants, there are no less than twelve. Under such circumstances one would think there should be a good opening in the provinces for a multitude of oculists. They would surely have working material enough, but if they had no means of their own they might starve. The greater part of the provincial population in Russia are either too poor to pay for medical services, or so much spoiled by the free medical help organizations of the governmennt, as to expect the same privilege from private physicians. Oculists of reputation, principally professors at the universities, have honored positions and lucrative practices. Aljantschikow estimates the number of eye patients to be 20 per cent. of the country population in Russia. Screbizki, in a report to the Curitorium of the Blind, states that contagious eye-disease is excessively widespread in Russia, and a large percentage of military conscripts have to be rejected every year on account of blindness or ophthalmic disease.

In the year 1893, on the initiative of Prof. Belljarminow at St. Petersburg, energetic steps were taken to relieve this calamity. A number of oculists were officially sent into the provinces to treat eye patients during one or several months at a time. Each oculist had a medical assistant, an orderly, and sisters of charity at his service, In the year '93 seven such expeditions were organized, by which 7691 patients were treated and 1416 operations performed, among which were 280 extractions of cataract. The results of these flying expeditions have been encouraging enough to repeat the experiment in the year 1894, when twenty-one expeditions were organized.

Though sceptically disposed, I wanted to satisfy myself by personal experience of the utility of this undertaking, and took a two-months' leave, which I spent in the department of Mariupol, government Jekaterinoslow, South Russia.

We were at work from May 13th to July 2d—that is, over fifty-one days, being at work nine or ten hours daily. During this time we treated 2154 patients, who made 4071 visits; 116 patients received hospital treatment. Among the operations we mention entropion 82, expression of trachoma 41, pterygium 22, iridectomy 58, cataract extraction 62. The number of incurably blind persons that came to the consultation was 120.

(The report goes into detailed descriptions of the different groups of disease and their treatment, operative as well as non-operative, for which we have no space.

Dr. Walter is of opinion that these "flying" expeditions, though expensive, are capable of doing a great deal of good. He endorses the proposition made by others, to appoint official oculists in the provinces, and to build for their use ophthalmic hospitals under government supervision. The oculists in charge should give accounts of their work, both charitable and scientific, in official annual reports.)

LIGHT—THE OINTMENT OF THE YELLOW OXIDE OF MERCURY—AND THE OINTMENT-POTS IN COMMON USE.

By S. HOLTH, of Drammen (Norway).

Translated by Charles H. May, M.D., New York.

THE ointment of the yellow oxide of mercury is acknowledged to be one of our most important and useful remedies. Though prescribed during the past century by Boerhaave, Richter, and A. Schmidt,[1] it is only during the present generation that it has come into extensive use; this was due to the elder Pagenstecher (Alexander), and the ointment generally bears his name.

It is a familiar fact that this ointment decomposes quite readily; a change takes place after a short period, sometimes at the end of a few days; as a result, there is grayish discoloration, which, becoming darker, finally changes to black. Altered in this manner, the ointment not only suffers in appearance, but though possibly harmless, it is less potent and may even be entirely useless.

If its use be limited to a few days, or if it be freshly prepared every day for use in clinics, there will be, practically, no decomposition; but as ordinarily prescribed to be applied at home for a considerable period of time, this change is an objectionable feature.

Though, undoubtedly, oculists have observed this tendency to decomposition, and some of them may have discovered the exact cause, ophthalmological and pharmaceutical works do not mention it, and erroneous views on the subject are quite prevalent.

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[1] J. Hirschberg, *Einführung in die Augenheilk.*, Leipzig, 1892, p. 18.

This prompted me to engage in a series of observations upon this subject. I noticed that discoloration first affected the upper surface and the portions in contact with the walls of the ointment-pot, and especially the *side exposed to light;* this indicated that the influence of light was a factor. An examination of the ointment-pots in general use showed that almost all of them, whether of milk-white glass, porcelain, or earthenware, were transparent to a greater or less degree,[1] and that the celluloid or wooden covers were translucent. Metal covers, though opaque, are not suitable since amalgamation with the oxide of mercury would result wherever the ointment came in contact.

I selected a number of ordinary porcelain ointment-pots with wooden covers, and coated the outer surface of both with a layer of black paint, so as to make them absolutely opaque. In a certain number, only two thirds of the circumference was painted, leaving one third free. In order to determine to what degree the influence of light was responsible for the decomposition and to what extent other causes, such as temperature, access of air, the particular ointment-base, etc., might operate, I proceeded in the following manner:

Five different portions of ointment were prepared, each containing two per cent. of the mercurial, but made with the following constituents:

1. Vaselinum flavum.
2. Vaselinum album.
3. Unguentum aquæ rosæ (cold cream).
4. Lanolini purissimi (Liebreich).
   Vaselini flavi........................āā. 1.
5. Lanolini purissimi (Liebreich).
   Aq. destill. coct......................āā. 1.
   Vaselini flavi.......................... 2.

Each variety of the ointment was subdivided and distrib uted among different kinds of receptacles: ordinary ointment-pots with various forms of covers, others which had been entirely or partially coated with a layer of black paint, others of colorless glass, and glass tubes sealed at both ends.[1] In this manner a large number of specimens were provided and distributed in various ways: (1) exposed to direct sunlight, (2) placed upon my desk, (3) left in a corner of the room which the sun never reached, and (4) kept in a perfectly dark room. Several glass pots and sealed glass tubes were put into a blackened cigar-box and placed within an ice-chest (8 ° C.). Others were exposed outside of the window; some of these were placed beneath a bell-jar, upon ice which was replaced constantly. Some were allowed to remain closed throughout the entire period of observation; others were opened daily and used in practice; some of those in the dark room were left open.

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[1] The old-fashioned, thick-walled ointment-pots made of clay or earthenware, with a depression near the margin of the opening to facilitate the fastening of the paper cover, are now scarcely ever used; these pots were often opaque, but, of course, the paper cover admitted light.

After five months of constant observation, I arrived at the following conclusions:

The ointment contained in the blackened pots remained unchanged throughout and of a fine yellow color, independent of the constituent, the place in which it had been kept, whether opened daily or kept open constantly. The ointment which had been prepared with cold cream rapidly became rancid and ill-smelling, but notwithstanding this change it remained of a nice yellow color.

On the other hand, the surface of the ointment preserved in the blackened pots showed a grayish discoloration *when exposed to light without covers;* this change took place in a couple of days in sunlight, and after several days in diffuse daylight; but the salve next to the walls of the pots was unaffected. *The blackened pots which were exposed without covers in the dark room or in the blackened cigar-box presented no change in contents in any part, including the surface.*

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[1] Unfortunately, I did not make use of colored glass pots. The following observation shows that red glass would have protected the salve from decomposition: A porcelain pot was filled with ointment and provided with a transparent, red celluloid cover. Discoloration took place along the walls of the pot, but the upper surface of the salve remained unchanged. When only half-filled, the upper surface also became discolored—due to the influence of non-colored light penetrating the transparent walls of the receptacle.

The contents of the pots which were only partially blackened showed changes in those portions only which corresponded to the transparent, unblackened areas.

In every case in which the ointment was exposed to sunlight, or to diffuse daylight in the common forms of pots and in glass pots, grayish or black discoloration followed in a shorter or longer period depending upon the intensity of the light, independent of whether the pots were provided with covers. *But when preserved in the dark, there was no change in the salve.*

When placed in ordinary pots, glass pots, and in sealed glass tubes, and kept on ice beneath a bell-jar (exposed to the sun), the ointment showed black discoloration in several days, independent of the constituent.

These results agree with the statements found in text-books of chemistry: That the red and yellow oxides of mercury are reduced through the influence of light. They justify the following conclusions:

1. Decomposition of the ointment of the yellow oxide of mercury, showing itself in grayish discoloration, is not due to chemical changes produced in the ointment base, but is *dependent entirely upon the reducing effect of light passing through the more or less transparent walls of the ointment-pots in ordinary use;* violet light probably exerts the most effect in this direction.
2. This decomposition is not influenced by temperatures between 0° and 30° C.
3. It is not caused by access of air.
4. Any ointment-pot may be used if kept in a dark place.
5. The use of absolutely opaque pots provided with similar covers, is the most practical way of avoiding decomposition.

The different pharmacopœiæ give directions regarding the method of preserving the yellow oxide of mercury; the larger pharmaceutical books, though they repeat these directions, do not mention anything concerning the influence of light upon the yellow oxide ointment nor the necessity of using opaque ointment-pots. With the exception of the work of Prof. M. K. Loewegren (Lund, Sweden), nothing is said on this subject by our two great treatises on ophthalmology[1] nor by ophthalmological text-books in general.

Regarding the ointment-base, I might add that cold-cream is, in one respect, the best constituent; on account of the great proportion of water which it contains, it distributes itself more readily over the inner surface of the lids than does any other base; but it becomes rancid very easily. Vaseline is suitable and absolutely unchangeable, but does not mix as readily with the conjunctival secretion as does cold cream. For these reasons I use a *modified cold cream* which spreads itself as readily over the parts as the ordinary form and does not become rancid; it is prepared as follows:

R	Hydrargyri oxidi flavi........................	0.05	0.20
	Lanolini purissimi (Liebreich) destill. filtrat. et Aq. coct.................................	aa	2.50
	Vaselini flavi.................................		5.00

M. D. ad ollam pellucidam cum operculo ligneo nigro.

Besides the yellow ointment, there are others used in ocular therapeutics, which are decomposed to a greater or less extent by the influence of light—for instance, the ointment of the white precipitate, the ointment of the red oxide of mercury (now superseded by the yellow oxide), and iodoform ointment (in which light causes the separation of free iodine and changes the preparation into a brown and irritating salve).

Hence, absolutely opaque ointment-pots should be used extensively; in fact, it would be well to employ them exclusively. The old-fashioned clay or earthenware ointment-pots would answer if provided with opaque covers; however, the inner surface of such vessels is often porous and difficult to cleanse. At my request, George Wenderoth, of Cassel, Germany, has manufactured opaque ointment-pots of porcelain; these are of a pretty dark-gray color, enamelled internally and externally, and provided either with a black celluloid cover or, as I prefer, with a cover of resin-free wood, polished and blackened externally.

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[1] Graefe-Saemisch, vol. iv., p. 125; Wecker-Landolt, vol. i., p. 348.

DIPLOPIA IN THE PERIPHERY OF THE FIELD OF FIXATION AND ITS BEARING ON THE DIAGNOSIS OF MUSCULAR PARALYSIS.

By Dr. A. DUANE.

I AM indebted to Dr. Alling for calling my attention both orally and also in his recent article in these ARCHIVES[1] to the frequency with which diplopia occurs normally in the outlying portions of the field of fixation. This is a point to which I had given too little thought before, and it is quite likely that in one or two cases in which I found diplopia in the upper portion of the field, I wrongly attributed this diplopia to an insufficiency of one of the levators, when actually no abnormal conditions at all were present. I cannot, however, admit that this fact invalidates in the least the diagnosis made of the majority of the cases which I reported in these ARCHIVES (January–April, 1894), and the statements and conclusions contained in that article I still regard as perfectly sound. My reasons for thinking so are as follows:

Dr. Alling's position may be summed up in the statement that diplopia of the kind observed in the cases cited occurs normally *when the eyes are carried nearly to the limits of the field of fixation*, and is due partly to the inability of one eye to keep up with the other when these limits are approached, partly to the effect of projection. That is, according to his view, a vertical diplopia, which makes its appearance when the eyes are directed far up and to the right, means one of two things. Either the right eye actually falls below the left in its upward movement—but does so simply because it has about reached the normal limit of its range of excursion and can naturally go no farther; or, the two eyes being on a level, the physiological divergence, which occurs in looking upwards, prevents both eyes from being accurately fixed upon the object, and the latter therefore appears double, the two images being heteronymous and having the same relation as though projected upon the surface of a sphere. In the latter case, he says *the inner image owing to the wheel-rotation of the eye must necessarily appear higher.*

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[1] July, 1895.

Now to certainly the greater part of my cases, these postulates, however correct they may be,[1] do not apply at all. For, so far from occurring only at the periphery of the field of fixation, the diplopia here began to appear when the eyes had moved but a little way from the primary position and increased very rapidly as the eyes were carried still farther from the latter. Thus taking the ten cases (out of the fifteen originally reported)[2] in regard to which I have been able to obtain positive data since the appearance of Dr. Alling's criticisms, I find that in Nos. 1, 7, 8, and 12 a noticeable vertical diplopia was already present in the horizontal plane and increased rapidly as the eyes were carried upwards. In case 11 vertical diplopia began 6° above the horizontal plane; and in case 2 it began when the eyes had been carried 20° out and 15° up, and was already 2° in amount when the eyes were carried 5° farther up. In case 14, vertical diplopia was present when the eyes were carried 25° out and 15° up. Again in case 1, the vertical diplopia (taken at a distance of 1 metre) finally attained 5 or 6° (equivalent to the deviation produced by a 12° prism); and in cases 7, 11, and 12 was almost or quite as marked. In one of the cases examined since, the vertical diplopia at an elevation of 30° was 3° and in still another amounted to no less than 8° (equivalent to the deviation produced by a 16° prism) at the same elevation. Moreover, the diplopia was not only large in amount and insuperable by voluntary effort, being in these respects altogether distinct from normal diplopia or that due to simple tiring of the muscles; but was also present by repeated tests and under all sorts of conditions. Thus the ordinary test with a candle at 1 metre was supplemented by that with a dot on a plane surface (held perpendicular to the line of sight so as to obviate the effects of false projection) and by tests made at a distance both with a stationary[1] and a moving test object. The results were uniformly concordant. Thus in cases 2, 7, 8, 9, 11, and 14, a vertical diplopia of the same character existed both for near points and for a distance of 15 or 20 feet. The same result has also been found in cases subsequently examined. This result, it need scarcely be noted, would not occur were the diplopia due simply to a transitory weakness of the muscles or to a normal inability of one eye to follow the other in its movements.

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[1] I say, "however correct they may be," for it can, I think, be demonstrated satisfactorily that Dr. Alling has fallen into two or three serious errors in his enunciations. As, however, the phenomena that he describes involve theoretical considerations of much intricacy and have no real bearing on the matters at issue, I will not take up space with their discussion.

[2] Cases 3, 4, 5, 6, and 10 could not be got at for re-examination, and the notes I have regarding them are not sufficiently precise to enable me to state with absolute certainty that in them the diplopia was greatly in excess of the normal in amount or that it occurred very far within the normal limits of the field of fixation. These cases, therefore, will have to be excluded entirely from consideration, in spite of my belief that two or three of them, at all events, were instances of the pathological condition in question. Their omission is the less to be regretted, since I am able to supply their places with at least the same number of additional cases in which the diagnosis was beyond a doubt.

Again, it will be seen that, according to the second of Dr. Alling's postulates, if lateral diplopia occurs in looking upward and to the right *the inner* (*i. e.*, *the left-hand*) *image must be the higher*. Indeed this follows at once from his figure, if we admit a wheel-rotation in the sense that he claims. And it will be always the left-hand image that is the higher, whether this image actually belongs to the right eye or to the left. Now, if the diplopia is heteronymous, it will be the image formed by the right eye that is the higher, and this, in fact, is what occurred in most of my cases. But in three or four cases, at least (including some not given in my former list), the diplopia was homonymous, and here, according to Dr. Alling's theory, the image formed by the left eye should have been the higher. This, however, was not the case, for here, too, the image formed by the right eye, *i. e.*, the *outer* image in this instance, was the higher. This result is quite inexplicable upon Dr. Alling's theory.

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[1] In this case, of course, the head was rotated and the eyes kept fixed. The advantage of this method of examining is that if the images of the object are projected against a vertical wall, the lines of sight always remain perpendicular to the latter, or plane of projection.

Quite as impossible of explanation upon the projection-hypothesis is the fact that the *vertical diplopia was often in excess of the lateral and was sometimes marked when there was no lateral diplopia at all.*[1] If the projection-hypothesis were correct, the vertical diplopia should vary directly as the lateral. In fact, as an examination of Dr. Alling's diagram will show, the ratio between the two should be expressed by the formula:

Vertical diplopia = lateral diplopia × tan α.

Where α = angle of rotation of the horizontal meridian of the retina according to Helmholtz. Now if the lateral diplopia becomes zero, the vertical diplopia should likewise be zero, which is by no means the case.

Moreover, as the greatest possible value of α (when the eyes are directed 45° up and 45° to the right) is 15° (Helmholtz), the tangent of which is 0.268, the vertical diplopia should at the very utmost be less than $\frac{1}{3}$ of the lateral.[2] In the same way, when the eyes are directed 30° up and 30° to the right, the vertical should be only $\frac{1}{7}$ of the lateral diplopia; and only $\frac{1}{14}$ of the latter, when the fixation lines are directed 30° to the right and 15° up. It is quite impossible, therefore, to explain by any such hypothesis as this, a vertical diplopia which, at even a relatively short distance above the horizontal plane, equals, or even exceeds, the vertical diplopia.

Furthermore Dr. Alling's hypothesis fails to explain *the*

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[1] Thus in case 11, the vertical diplopia, when the eyes were directed up and to the right was $3\frac{1}{2}°$ (= deviation produced by a 7° prism), while the lateral diplopia was 0°.

[2] In convergent positions of the eyes the amount of vertical as compared with the lateral diplopia is slightly increased by the fact that, when the gaze is directed up and to the right, the object of fixation is somewhat nearer the right eye. When, however, as in my cases, the observations were made at a distance of three feet at the lowest, the increase of vertical diplopia due to this fact becomes very small, so that even in the extreme oblique positions the total amount of the vertical is still not so great as one third of the lateral diplopia; and in positions less removed from the primary, this increase is practically infinitesimal.

markedly unilateral character of the phenomena in many of the cases. He himself endeavors to account for this by supposing that the head was tilted or that a hyperphoria was present, making one image naturally higher than the other. But the discrepancy was much too great to be met by the former explanation. Tilting of the head does not cause a very great displacement of the position of the images, even when carried quite far; and it was not carried far enough to be noticeable in any of the cases that I tested and which I carefully watched to obviate this very source of error. It would be quite futile, for example, to try to explain in this way a case in which, as in one of mine, one image of an object three feet distant was five inches higher than the other, when the eyes were directed up and to the left, but were on a level when the eyes were directed up and to the right.

The hypothesis of a pre-existing hyperphoria is equally inapplicable. For the hyperphoria must either have been variable in amount, or constant in all parts of the field of view. If the former, it argues at once a relative weakness of one of the elevators of the eye, *i. e.*, the very condition that we are contending for; for a hyperphoria which decreases to nothing below the horizontal plane and increases steadily above can be accounted for in no other way. If, however, Dr. Alling means by hyperphoria an elevation of one eye above the other of constant amount (concomitant deviation), this, like all other concomitant deviations, would be equally marked in all parts of the field of fixation, and be as evident below the horizontal plane as above it. This, however, was not the case in any one of the patients I examined. The unilateral character of the phenomena was marked in cases 1, 7, 8, 12, and 13, and in all of these the discrepancy between the amount of vertical diplopia obtained in the right oblique position and that obtained in the left was such as to be quite inexplicable upon either of Dr. Alling's hypotheses.

Finally, a word as regards the *tilting* of the double images. Dr. Alling finds the explanation of this phenomenon in Meissner's experiments proving the existence of a divergence of the vertical meridians when the eyes were converged and directed upward. Dr. Alling forgets apparently that

Meissner's experiments (which, by the way, were performed independently and with much more precision by LeConte) were made with strong convergence of the eyes. In the cases which I reported the tilting of the images was observed when the eyes were either but slightly converged (object at 1 metre distance), or when there was no convergence at all (object at 15 feet). In case 11 the angle of divergence of the double images at 15 feet (*i. e.*, their deviation from parallelism) was at least 10°.

My own conclusions, drawn from a large number of examinations of the field of fixation and of diplopia occurring in all parts of the field, are as follows:

1. Diplopia occurring at the limits of the field of fixation (beyond 45° from the primary position) is physiological. It is not, however, by any means always present even there, and in any case is slight in amount, and in the same case often variable and inconstant. *It is solely diplopia of this sort to which Dr. Alling has reference in his article and to which his arguments apply.* It may be due to a natural failure of one eye to keep up with the other in its excursion, or to the effects of projection, being in the latter case brought about by the rotation of the retinal horizon, real or apparent, that occurs in oblique positions of the gaze. But whether projection can produce any diplopia at all of the kind that I found in my cases is doubtful, and in any case the effect of projection in producing vertical diplopia is very slight and makes itself apparent only at the limits of the field of fixation.

2. In a large number (probably the majority) of persons with natural eyes it can be proved that binocular single vision is still present even when the eyes are carried further than 45° from the primary position.

3. Well-marked diplopia occurring as a constant phenomenon within 40° of the primary position indicates a weakness or at least a want of balance of the eye-muscles. If this diplopia is still quite peripherally located, *i. e.*, occurs not less than 30° from the primary position, it indicates only a slight and, very likely, temporary muscular disability, such as may occur in neurasthenia, etc. As weakness from

this cause is very apt to be symmetrical, the diplopia is often present to about equal extent in all the oblique positions of the gaze, both to the right and left, and above and below.

4. A diplopia which begins to be apparent near the primary position and increases rapidly as the eyes are carried in any one direction indicates a serious impairment of muscular energy, *i. e.*, a true paresis. The diagnosis will be confirmed if upon repeated examinations it is found to be constantly present and particularly if it shows a markedly unilateral character. *Diplopia of this sort Dr. Alling does not consider in his article*, and to it, therefore, his arguments do not apply. As eight out of ten of the cases I originally described, as well as some six cases which I have examined since, belong to this category, I must adhere to my original diagnosis and to the opinions based upon it.

REPORT OF THE TRANSACTIONS OF THE TWENTY-FOURTH OPHTHALMOLOGICAL CONGRESS, HELD IN HEIDELBERG, AUG. 4 TO 7, 1895.

By Prof. **HORSTMANN** and Dr. **GREEFF** (Berlin).

Translated by Dr. C. Zimmermann, Milwaukee, Wis.

About 120 members and guests were present. In the evening of August 4th, reception and social meeting took place at the Stadt-Garten. After a short address by Prof. von Zehender, August 5th, 9 A.M., the transactions commenced at the aula of the university.

I. Sitting: Monday, August 5th.

President: Mr. A. Robertson (Edinburgh).

1. E. Fuchs (Vienna): (a) *On erythropsia.*

Fuchs examined on himself and others the erythropsia occurring when going over snow-fields. The dazzling caused by the snow need not be painful, but must continue for a long while. An elevation of 1500 metres above the level of the sea is necessary. In a snow-hut, Fuchs, with his assistant, noticed after a long march over the snow, that at first the black squares of a chessboard appeared green, and after about fifteen seconds the white squares also. Then a pink stripe commenced to appear at the margin of the white squares, which soon became enlarged. After two to three minutes this phenomenon disappeared. The erythropsia has an extension of about 10–20° towards the periphery of the visual field, and is mostly more intensive and extensive in the upper portion of the periphery, which is dazzled more by the snow. The erythropsia occurs in a zonula around the centre; the latter is colorless.

In aphakia, dazzling by snow at once elicits erythropsia. In general it is due to the glare, and occurs in the retina, because it can only be observed in the one eye, left open, after closure of the other. Two explanations are possible: either erythropsia is due to an irritation of the retinal fibres conveying the perception of red or to the becoming visible of the eye's visual purple, which is in the state of regeneration. The latter Fuch thinks to be the more probable.

In the discussion GREEFF relates a case of spontaneous erythropsia, which was nothing but a mere monochromasia. Only dark- and light-red were seen.

HAAB and DUFOUR also took part in the discussion.

(b) *Minor communications.*

Fuchs explains peculiar cases of atrophy of the retina and choroid resembling the so-called retinitis pigmentosa, but causing large white atrophic patches. Colman Cutler described such cases of Fuchs's clinic in *Archiv f. Augenh.*, vol. XXX., THESE ARCHIVES, XXIV., 3.

2. A. VON HIPPEL (Halle): *The operative treatment of myopia.*

One of the most important chapters of modern ophthalmology is the question as to the justification of the operative treatment of myopia. Von Hippel operated about 60 cases of high myopia during the last years, which he recently re-examined, in regard to the final result. At first only young individuals were operated on, finally, also people up to fifty-five years. The sclerosing process in the lens does not occur in myopic eyes. There was no nucleus in the lens of a man fifty-five years old. The indication for the operation must not rest on the number of dioptries; in children the operation was performed from ten dioptries upward. About 20 cases had choroiditic foci or retinal hemorrhages, in which the operation turned out favorably. In order to obtain binocular vision the operation of both eyes is required. In one case iritis set in. One eye was lost by croupous conjunctivitis. Detachment of the retina was never observed after the operation. The difference of refraction was 14–24 D, *i. e.*, in myopia the lens has a higher refractive power. V was improved 4–10 fold. Von Hippel warmly recommends the operative treatment of high myopia.

3. H. SATTLER (Leipsic): The same subject.

Sattler operated on 85 highly myopic eyes in 68 patients since the commencement of 1893. He obtained final results in 65 eyes, of which the lowest degree of myopia was 9. D. In this case the operation was performed to impede the progress of myopia through school-influences. Most patients had 15–20 D myopia. Sattler thinks now, that cases of less than 12.00 D ought not to be operated on. The difference of refraction was 11.50–20.00 D; the average 15–15.50 D. V increased 3–4 fold, but not more. In a series of cases binocular vision was obtained by a binocular operation, which, therefore ought to be the rule. The patients operated on were from six to sixty-two years old. Sattler also found no nucleus in the lens of myopic eyes. He observed detatchment of the retina in 4 cases after the operation. The first case has to be excluded being of traumatic origin. In the second total detachment, after good sight had existed for four weeks. In cases 3 and 4, both eyes had been operated upon; one eye remained good, in the other detachment occurred. Sattler does not see a contra-indication against the operation in the detachment, as this may happen in myopic eyes also without operation.

The papers 2 and 3 gave rise to a lively discussion. FUCHS relates the case of a young man who had been operated on for zonular cataract nine years ago. His myopia turned into emmetropia by aphakia. Then the myopia slowly increased.

SCHWEIGGER in general agrees with both speakers. Emmetropia or slight hypermetropia after the operation has to be aimed at. Hypermetropia of a higher degree, even from 4 D on, is very disagreeable. Schweigger operates, when the far point of the eye lies in 7 *cm* (= 15–16 D) or nearer. The refraction is best measured objectively with the electric ophthalmoscope devised by him.

In the discussion take part: Ostwalt, Rosenmeyer, Thier, Schön, Elschnig, and Haab.

4. UHTHOFF (Marburg): *On the pathological anatomy of purulent keratitis* (investigations made in conjunction with Dr. Axenfeld).

Uhthoff and Axenfeld had an opportunity to examine microscopically eleven human eyes affected with purulent keratitis, four cases of serpent corneal ulcer, characterized by spreading in the surface (progressive borders), while the portions diseased at first were again covered by epithelium, three cases of keratomalacia, *i. e.*, hypopyon-keratitis of the portion corresponding to the palpebral fissure, rapidly spreading into the depth, chiefly met with in small children, and severe general cachexia, then three cases of ulcus serpens starting a panophthalmitis, and a case of binocular neuroparalytic keratitis in locomotor ataxia, which is very similar to kerato-malacia. All these forms of hypopyon-keratitis show that the principles arrived at by Leber in keratitis of rabbits also hold good for man, although some questions, *e. g.* in regard to the immigration-ring, could not be determined any more on account of the generally far advanced stage. Especially it could be ascertained that the hypopyon derived its origin from the iris and the sinus of the anterior chamber. The circumscribed perforation of Descemet's membrane, set forth by Verdese and Fuchs, before the general rupture of the cornea, could not be observed. Uhthoff warns against confounding it with places in which the membrane, struck obliquely, is only covered by cells. With the aid of a large number of drawings the changes of the endothelium, the substance proper, etc., are minutely discussed, and pictures of different kinds of pneumococci shown, the chief producers of the serpent corneal ulcer according to the investigations of Uhthoff and Axenfeld. The non-serpiginous forms have a different bacteriology.

Discussion : Wagenmann.

5. TH. LEBER (Heidelberg) : *Case of conjunctivitis petrificans.*

Leber observed a new and rare disease of the conjunctiva. A similar case was sent to him for examination by von Hippel. The conjunctiva is the seat of an inflammatory swelling with white opaque spots gradually increasing in size and becoming confluent, until they form a calcareous mass hard as stone which consists of calcareous infiltration of the conjunctiva.

VOSSIUS and BRÜCKNER relate similar cases.

6. A. VOSSIUS (Giessen) : *A case of lipoma of the upper lid.*

Lipoma of the upper lid is not as rare as asserted in literature. Only a few cases are described. Mooren reported 24 cases, among which some were apparently confounded with ptosis adiposa. Vossius saw a sharply defined tumor on the inner half of the upper lid of a man fifty-six years of age, at a place where the palpebral cysts are generally situated. A periosteal sarcoma was thought of. The tumor could easily be enucleated. At its base, where it adhered to the periosteum, a plexus of nerves was found. It contained white cheesy masses like a dermoid cyst. Microscopically it was a pure lipoma.

Discussion : LANGE. A. VON HIPPEL (Sen.) saw a boy of seven years with a lipoma on the upper lid the size of an apple.

7. J. MICHEL (Würzburg) : *The decussation of the nerve fibres in the chiasma.*

Michel is sorry that scarcely any attention has been paid to his opinion of the total decussation of the nerve fibres in the chiasma. To prove the decussation he used two methods: (1.) Artificial atrophy of one optic nerve; (2.) series of sections through a normal chiasma, stained with Weigert's method. He demonstrated his preparations to Koelliker, who also made new series of sections through the chiasma. Koelliker corroborates that, despite the most careful scrutiny, no non-decussating fascicle could be found. If there are non-decussating fibres at all, they certainly are so scarce that they are physiologically of no account, and may be taken for the centrifugal optic fibres according to Ramon y Cajal.

8. L. Bach (Würzburg): *Experiments on the etiology of sympathetic ophthalmia, with demonstrations.*

Numerous experiments with staphylococci of different virulence, pneumococci, and tubercle bacilli, proved that a migration of bacteria from the first to the second eye along the optic sheaths does not occur in the sense of the migration theory.

The bacteriological examination (made according to the most diverse methods) of optic nerves and eyes which had been enucleated on account of imminent or actual sympathetic ophthalmia, always was negative.

After resection of a piece of the optic nerve, there never was a communication re-established between the vaginal spaces of the central and peripheral optic stumps. Through different kinds of irritation of the ciliary nerves, changes in the second eye were noticeable in half an hour. They were to be considered as the first signs of sympathetic inflammation. They consisted in the formation of fibrine and a focal congeries of corpuscular blood elements in the anterior chamber, on the anterior surface of the iris, between the ciliary processes in the peripheral portions of the vitreous body, and between choroid and retina. In two experiments a vesicular detachment of the epithelium of the ciliary processes took place; the vesicles contained fibrinous coagulations.

Bach thinks the transmission to be thus: From the first eye the irritation is propagated through the ciliary nerves to the ganglion ciliare, through its sympathetic root to the carotic plexus of the sympathetic nerve of the same side, then through the circulus arteriosus Willisii to the carotic plexus of the sympathetic of the other side, and then in a centrifugal direction to the ciliary ganglion and the ciliary nerves of the second eye. The transfer of the irritation may be effected directly by the vaso-dilatory sympathetic fibres of the trifacial nerve as well as indirectly by reflex action from the sensitive fibres of the latter, the irritation irradiating in the medulla oblongata from one side to the other.

II. Demonstration-Sitting: Monday, August 5th, at 2.30 P.M.

Chairman: A. VOSSIUS.

1. L. BACH (Würzburg) demonstrates:

(a) *The nervous plexus in the eyelids* according to Golgi-Cajal's method.

(b) *Human retina* according to Cajal.

(c) *Hydrophthalmus in rabbits resulting from inflammation in fœtal life.*

2. W. VON ZEHENDER (Munich) explains and demonstrates his *goniometer*, an instrument for the exact measurement of the degree of strabismus.

3. W. UHTHOFF (Marburg) shows the *preparations* belonging to his paper.

4. TH. LEBER (Heidelberg):

(a) Presents the patient with *conjunctivitis petrificans.*

(b) Demonstrates *preparation of a teratoma of the cornea*, containing acinous glands and cartilage. Leber thinks that the cartilage originated in the semilunar fold. Anterior and posterior synechiæ; undoubtedly a perforation of the cornea during fœtal life had occurred.

(c) Demonstrates *preparations of congenital cataract in a rabbit.*

5. A. VOSSIUS (Giessen) shows the *preparations* illustrating his paper.

6. OSTWALT (Paris) demonstrates:

(a) An *ophthalmophorometer.* This instrument, with the aid of Maddox's rod, enables us to readily determine in a few minutes the state of equilibrium of the eyes in a horizontal and vertical direction, for distance (5 *m*) and near-by ($\frac{1}{3}$ *m*).

(b) A *pupillo-statometer*, admitting of rapid measurement of the pupillary distance, even in strabismus.

7. SULZER speaks on *artificial replacement of the cornea.* Sulzer made experiments, based on Leber's investigations on inflammation, to find a prothesis for total opacity of the cornea. Most

substances are soluble in animal fluids, platina and pebble least. Sulzer attaches a lentiform piece of pebble, mounted in platina, to the posterior surface of the cornea with wires and barbed hooks. The operation is not very difficult, and the piece heals in well. This done, the overlying portion of untransparent cornea is excised with the trephine. Sulzer shows a rabbit wearing the prothesis for a year.

8. A. ROBERTSON (Edinburgh) shows *preparations of filaria* from the eye of a patient who had lived in West Africa for several years.

9. HOVI (Tokio) shows his *ophthalmo-skiascope.*

III. Sitting : Tuesday, August 6th, at 9 A.M.

Chairman : E. FUCHS, Vienna.

1. RINDFLEISCH (Weimar) reports a case of *isolated left-sided paralysis of the sphincter iridis*, which he thinks to be nuclear. After half a year it was followed by monocular reflex immobility of the pupil, with partial paralysis of accommodation in the same eye.

2. TH. LEBER (Heidelberg) : On the *change of fluids in the anterior chamber.*

This cannot be seen, as the fluids are clear like water. Staining them with cyanate of potash or fluorescin has been tried. These methods, however, do not show that there is a current, as the molecules of the substances only diffuse, the water itself remaining quiet. The diffusion of these substances does not indicate, therefore, the course the fluids take. The quantity of the fluid flowing through the anterior chamber can be best ascertained, if in a dead eye the quantity of fluid, filtrating through the sinus outward, is determined.

Leber emphasizes specially that there is no current in the anterior chamber, but only a very slow change of fluids. He could observe this in two living individuals. The aqueous is renewed about twice within one hour.

3. E. VON HIPPEL, Jr. (Heidelberg) : *On parenchymatous keratitis.* Von Hippel gives the results of the clinical examinations of 80 cases of parenchymatous keratitis, making use of the incident literature.

Hereditary syphilis plays a very important part, but is not the only etiological factor.

Especial attention has to be paid to tuberculosis as an etiological factor, which von Hippel studied very carefully in connection with a former case, which he examined anatomically.

In 28 cases there was marked tuberculous taint, *i. e.*, objective symptoms of tuberculosis could be ascertained.

Fifteen of these 28 cases showed simultaneously clear or uncertain signs of syphilis, which were wanting in 13.

Von Hippel reached the conclusion that, with our present knowledge, we are justified in making the diagnosis of tuberculous parenchymatous keratitis with the greatest probability, if characteristic signs of lues are wanting, but tuberculous predisposition or objective tuberculous symptoms are present.

The possibility is alluded to that the eye-disease may be of tuberculous nature, lues co-existing at the same time. The question, whether there are certain objective symptoms of the eye-disease that would prove the syphilitic or tuberculous character, has to be answered in the negative. The remaining vessels in the deeper strata of the cornea and the choroiditic changes, considered by Hirschberg as characteristic of lues, are also found in the tuberculous form. Formation of nodules in iris, sinus of anterior chamber, and cornea were mostly found in the tuberculous form, four times in persons who were syphilitic almost beyond doubt. No treatment can prevent the outbreak of the disease on the second day of the treatment. A general mercurial treatment is urgently indicated in syphilitic parenchymatous keratitis. Hochsinger observed, that of 63 individuals with undoubtedly hereditary syphilis, who were subjected to an energetic anti-syphilitic cure at their early age and were under observation for 4–22 years, not one suffered from parenchymatous keratitis, Hutchinson's teeth, or deafness.

Discussion : Michel, Wagenmann, Manz, Franke, Uhthoff ; Vossius also emphasizes that the hereditary lues does not play such an essential part in parenchymatous keratitis as it was generally believed ; sometimes it is caused by rheumatism.

4. AXENFELD (Marburg) : *On the so-called anterior ciliary nerves.* The terms, "long," "short," "direct," or "indirect," ciliary nerves do not mean that the long (direct) ones enter the sclera in front, but only that they do not pass through the ganglion. Therefore the latter do not furnish an explanation of the sometimes insufficient result of neurotomy of the ciliary nerves on the posterior surface of the globe. The "nerfs ciliaires superficiels" of Bou-

cheron (inserting in front) innervate only the surface of the corneal periphery. In regard to other "anterior" ciliary nerves, *i. e.*, such as pass in front through the sclerotic to the uvea, etc., anatomists widely disagree. According to Axenfeld's preparations and series of microscopical sections, there are:

(a) Several episcleral relatively thick twigs of the ciliary nerves running to the front, the relations of which to the interior of the eye are as yet doubtful. They are cut by neurotomy, as they lie in the path of the other nerves as far as the sclerotic.

(b) Very peculiar heretofore unknown nerves perforating the sclerotic vertically, the nature and origin of which could not be elucidated.

The failure of neurotomy may be explained by abnormal varieties of the entering nerves, as shown in the atlas of Svitzer and a preparation of Axenfeld. The operation from the medial side is to be preferred, as well as resection to simple neurotomy.

5. KRUEKMANN (Leipzig): *Test of sensibility of the cornea.*

Kruekmann shows Fey's "irritating hairs, which he tried on diseased eyes in regard to the sensibility of the cornea. He illustrates their value especially in glaucoma and herpes of the cornea. The examination with the irritating hairs is a safe and quantitative method for determining any disturbance of corneal sensibility, no matter what its cause. Through this method Von Fey's opinion was corroborated, that the cornea and conjunctiva lack the sense of pressure and show only sensation of temperature and a general and very keen sensation of pain. The former is confined to the conjunctiva and the marginal portions of the cornea.

Then the action of mydriatics and myotics on the intra-ocular pressure was tested, with the following result: The myotics at first increase the pressure and then lower it, the mydriatics *vice versa.* The disturbances of sensibility in locomotor ataxia, ascertained by this method, help the diagnosis of the paræsthesiæ of the fifth nerve, which are sometimes the precursors of tabes.

6. ZIMMERMANN (Jena): *Ulcus serpens.*

Zimmermann gives statistics of 60 cases of ulcus serpens treated with galvano-cautery at the clinic at Jena. He describes the procedure, as it is practised in nearly all clinics. Nothing essentially new.

Discussion: SCHIRMER says, that the pain in keratitis is very different according to the seat of the affection. In cases of dacryocysto-blennorrhœa, extirpation of the lachrymal sac ought to precede the treatment of the ulcer.

Nieden (Bochum) found the galvano-cauterization of the ulcer very useful in more than 2000 cases. In the further discussion take part: Schloesser, Kalt, Bach, von Hoffmann, Thier.

7. Otto (Leipzig): *Changes of the fundus oculi in highly myopic eyes.*

Staphyloma posticum is generally not considered to be an ectasia of the background of the eye. Weiss saw among 20,000 eyes only 2 ectasiæ, which he took for anomalies of primary formation. Caspar, who saw them more frequently, thinks them to be congenital and related to the ocular fissure. Otto found in the eye clinic at Leipzig trough-like ectasiæ in the fundus oculi of numerous myopes. At the margin of the ectasiæ dark shadows are visible, and the difference of level can be proven by parallax. The shadow is best visible through narrow pupils. Sometimes within a large ectasia there was another, smaller one. It may extend from the optic nerve beyond the macula as far as the region of the vorticose veins.

Leber, in the discussion, pointed out that more than thirty years ago, in von Graefe's clinic, the distinction was made of staphyloma posticum verum, with ectasia. Weiss observed 5 further cases of ectasia of the fundus; in one case it was annular.

8. Elschnig (Graz): *Remarks on glaucomatous excavation.*

It is generally agreed upon that every glaucomatous excavation is due to pressure. Schnabel proved on two eyes with glaucomatous hypertony that glaucomatous excavation can occur after atrophy of the intraretinal and intrachoroidal portion of the optic nerve, the thin lamina being at its proper place. This case, however, was invalidated with the assertion that the diagnosis was erroneous, and that it was simply a case of physiological excavation. Elschnig gives the *post mortem* of an eye, which for four and a half years showed typical glaucomatous excavation, the anatomical examination of which corresponded perfectly to Schnabel's descriptions: glaucomatous, *i. e.*, steep marginal excavation, the lamina being in its normal position. With regard to the above-mentioned controversy in Schnabel's cases, the differential diagnosis of glaucomatous and atrophic excavations is discussed in detail, and it is pointed out that atrophy of the optic nerve cannot produce a steep marginal excavation, because only the nerve fibres, not the perineural tissue, waste. The aspect of an atrophic disc with pre-existing large physiological excavation may resemble that of glaucoma, but a careful examination will guard against errors.

There is, however, a glaucomatous excavation which is not due to pressure. It originates in the glaucomatous affection of the optic nerve. This is (according to the investigations of Schnabel, Schmidt-Rimpler, Fuchs, and Elschnig on eyes enucleated for inflammatory glaucoma, and according to the functional tests of glaucomatous eyes): interstitial neuritis of the portion of the optic nerve provided with a myeline covering, in the portion divested of their myeline sheath, either neuritis or usually simple atrophy of the entire intraretinal and intrachoroidal piece, appearing as glaucomatous excavation.

9. GROENOUW (Breslau): *Treatment of glaucoma.*

Groenouw warns, after the diagnosis of glaucoma is established, against the too prolonged use of eserine, as recently recommended especially by H. Cohn. He mentions cases, in which the glaucomatous process advanced despite daily instillations of eserine. The latter is only a palliative, no remedy. If a remedy, as Cohn communicates, requires daily use for fourteen years, it proves that the drug does not heal the process, and only abates each constantly recurring attack.

In the discussion to 7 and 8, SCHWEIGGER states, that there is no typical ophthalmoscopic picture of excavation by pressure. Even in physiological excavation the margin of the excavation may coincide with that of the disc. Therefore still other symptoms have to be considered for the diagnosis of glaucoma. As soon as glaucoma has become manifest, he thinks iridectomy indicated. In the discussion also take part: Schoen, Dufour, and Michel.

IV. Demonstration-Sitting: Tuesday, August 6th, 2.30 P.M.

Chairman: O. HAAB (Zurich).

1. SNELLEN (Utrecht) shows *color phenomena on a rotating disc.* If a black disc with a white sector is turned from right to left, the black prevails, *vice versa* the white, because in the first case the after-image of black encroaches upon white, with the reverse in the second case. In England a toy is constructed according to this principle. Simultaneously color phenomena appear during rotation.

2. O. SCHIRMER (Greifswald) shows his *new pupillometer.*

3. R. GREEFF (Berlin) reads a paper on the *histology and pathology of glioma retinæ*, with demonstrations.

A section through a glioma retinæ, highly magnified, always shows various kinds of cells. From this it may be inferred that it does not consist of a single species of cells. If it contains glia cells, as Virchow and many other authors assume, they must be brought in view in such perfect shape by means of Golgi-Cajal's method, as can be done with brain, optic nerve, and normal retina. In this manner Greeff attained very nice impregnations of several recent gliomata. The impregnated cells cannot be anything else but of a nervous or gliomatous kind, as other cells cannot be displayed by this method. The tumor consists of neuroglia cells with an enormous number of fine, long processes, often entangled in a thick plexus. The cells resemble the embryonal spider cells in the optic nerve, as described by Retzius. Between these there are other cells, entirely different, with sharply defined cell-body and thick processes, that spread dichotomously. Their form and size vary greatly. They are ganglion cells as met with in thick tumors, although rarer than the neuroglia cells, and so nicely preserved as not to be considered as accidentally immigrated into the tumor from the normal retinæ.

Then there are small cells and fibres with nuclei, resembling very much the forms of development of nervous cells. The formation of glioma probably takes place from displaced germs in the retina, *i. e.*, accumulations of cells, which in the embryonal retina were dislodged from one stratum into the other. As this may occur from any stratum into any other, glioma may grow from any stratum of the retina.

Glioma therefore is a tumor due to a malformation in the retina. Essentially it consists of exuberating glia cells, forming a thick plexiform filament with their processes. Among these the ganglion-cells are imbedded.

4. WINTERSTEINER (Vienna): *On corneal alterations in neuro-epithelioma (glioma) of the retina, with demonstrations.*

Wintersteiner reports corneal changes he found in 32 cases of neuro-epithelioma of the retina, with the following classification:

(1) Such, as caused by glaucoma, or the expansion of the walls of the globe. To this category belong circumscribed atrophies by stretching or ruptures of Descemet's or Bowman's membrane. The former resists longer; its elasticity and endurance in keratocele are known. It also exhibits signs of globular new formation at the margin of the ruptures, whereas Bowman's membrane cannot be replaced. Wintersteiner found cystic cavities in the basis substance, lined with cells similar to endothelium.

(2) Changes by immigration of elements of the tumor, causing atrophy of the parenchyma and other parts. In our preparation were cells of the tumor in a newly formed corneal vessel, apparently owing to a metastasis.

(3) Changes by lagophthalmus from protrusion of the eyeball. A rapid suppuration takes place which is the most frequent cause of perforation of the globe. (This will be discussed more in detail in a monograph soon to be published.)

5. A. WAGENMANN (Jena) : *On foreign-body giant cells.*

Giant cells may be encountered in quite a number of diseases, especially around foreign bodies, including parts derived from the body of the patient. Wagenmann found giant cells around hairs, foreign bodies, in chalazia, around cysticerci, etc. They may also be evoked by cholesterine crystals. Recently Wagenmann observed giant cells around a spontaneously dislocated old cataract.

6. E. VON HIPPEL, Jr. (Heidelberg) :

(a) *On spontaneous resorption of senile cataract.* Demonstration. The nucleus of the lens of an individual aged sixty-nine was extracted within the capsule, the corticalis being totally absorbed. The anatomical examination revealed almost total absence of the capsular epithelium, just as in Mitvalsky's case. The loss of epithelium, from unknown causes, undoubtedly favors the transit of aqueous through the capsule and the absorption of the corticalis.

(b) Von Hippel presents a patient with an unusual affection of the retina, who has been under observation for two years ; a diagnosis, however, could not yet be made. He has tuberculosis of the right calcaneus, and had right-sided iritis and otitis media. Whether the retinal affection is due to tuberculosis, cannot be ascertained. The case will be further observed and more fully described later on.

7. L. WEISS (Mannhein) : *Demonstrates a binocular exophthalmometer.*

V. Sitting : Wednesday, August 7th, 9 A.M.

Chairman : DOR.

1. OSTWALT (Paris) : *Gumma of the ciliary body.*

In a man, aged thirty-nine years, a gumma developed in the ciliary body of the right eye, fourteen years after syphilitic infection, in the upper-outer region, not as usual in a radiating, but in an oblique direction, growing into the anterior chamber from its sinus. Three yellow-white puriform nodules successively advanced between iris and cornea, and gradually coalesced, under glaucomatous pain. At the same time the gumma extended into the sclera at the limbus, causing a lenticular prominence. Under sublimate injections and iodide of potash the tumor at first grew rapidly, but the intraocular tension, slightly increased, soon became normal. The tumor decayed, a pseudo-hypopyon formed from the detritus, and the whole mass of the tumor at once disappeared from the anterior chamber.

There was occlusion of the pupil from the accompanying severe iritis and reduced perception of light, which, however, after $3\frac{1}{2}$ or 4 weeks improved to $\frac{6}{10}-\frac{6}{8}$.

Oswalt thinks, that the sudden increase in volume of an intraocular gumma, with restitution of increased tension to a normal degree without perforation, is owing to an imbibition of the tumor, and gives a good prognosis, indicating the imminent decay of the tumor.

2. Hori (Tokio) : *Ophthalmia hepatica.*

Hori directs the attention to the connection of ocular affections with diseases of the liver. He gives the clinical history and histological and anatomical conditions of a man suffering from cirrhosis of the liver, associated with retinitis (changes of the intra-ocular vessels, tessellated intra-vascular spaces).

In the discussion Leber sets forth the intimate relation between hemeralopia and diseases of the liver, mostly associated with icterus.

Weiss found in such cases peripherally light spots in the retina ; Meyer, in Europeans who had lived in the tropics for a long time, also black pigment dots.

Uhthoff calls to mind hemeralopia in alcoholics. The affection of the liver most likely is the connecting link.

3. O. Haab (Zürich) : *Extraction of pieces of iron from the eyeball.*

Haab from continued experience thinks that his method, made known three years ago, is the best. He employs a magnet as strong as possible. The strongest magnet will yield the best results. For that purpose a special instrument has been constructed in Zürich which perhaps may be still improved. The small magnet of Hirschberg never will be entirely discarded ; it is, as it were, the best forceps for foreign bodies, if the magnet can be brought in contact with the piece of iron, but it does not act at a distance.

In 60 % of all cases of injury with chips of iron Haab attained useful vision.

Wagenmann, Seggel, Thier, Schloesser, and Leber participate in the lively discussion.

4. KALT (Paris) : *Treatment of purulent conjunctivitis.*

Kalt uses a small glass funnel for washing the pus from the conjunctival sac, which is pushed under the lids, without touching the cornea. An irrigator is attached to the funnel. In blennorrhoic conjunctivitis he irrigates the conjunctival sac with 1 to 2 quarts of a solution of permanganate of potash 1 : 3000 (one teaspoonful of a saturated solution to one quart of water) three times a day. In blennorrhœa brushing the lids with nitrate of silver may also be done, but is not necessary.

The blennorrhœa disappears on the 12th day. This treatment is also good in ulcus serpens and granular conjunctivitis. The level of the water in the irrigator must not exceed 30 – 40 *cm* above the eye. If raised higher, the patient experiences pain.

5. SCHWARZ (Leipzig) : *On hysterical amblyopia.*

Schwarz discusses the various forms of hysterical amblyopia (excluding the typical hysterical amblyopia) in the light of the somewhat altered definition of hysteria by Moebius. "All those pathological changes of the body and its functions, that are caused by psychical processes, are hysterical." He offers reasons for referring the above-mentioned forms of disease to hysteria.

Discussion : von Hippel, Jr., and Axenfeld.

6. L. WEISS (Mannheim) : *On the growth of the eyes.*

Weiss measured accurately the eyes of new-born children and of adults at different ages, and found that the volume of the eyes increases 3 or 4 fold during lifetime, the volume of the whole body 21 fold. Curves indicate that the eye grows at the same rate as the brain, equally in all its diameters.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FIRST QUARTER OF THE YEAR 1895.

By Dr. ST. BERNHEIMER, in Vienna; Prof. C. HORSTMANN, in Berlin; and Dr. P. SILEX, in Berlin;

WITH THE ASSISTANCE OF

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Metz; Dr. Sulzer, Geneva; Dr. Werner, Dublin; Dr. C. H.
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Ole Bull, Christiania; Dr. R. Greeff,
Berlin; Dr. Deus, Berlin; and
others.

Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. ST. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS. STATISTICAL REPORTS.

1. Rochon-Duvigneaud. A description of the normal anatomy of the eye, pp. 136, 23 figures. Paris, *Soc. d'éditions scientifiques*, 1895.

2. Pansier. The history of artificial eyes. *Ann. d'Ocul.*, cxiii., p. 112.

3. Pepljaschin. Surgical ophthalmology as observed in foreign eye clinics. *Wratsch*, 1895, No. 2.

4. Helmholtz. Handbook of physiological optics. 2d edition, parts 9–10. Hamburg and Leipsic, 1895.

5. SCHULEK. Hungarian contributions to ophthalmology. 1 vol. with 45 figures in the text. Deutike, Leipsic and Vienna, 1895.

6. MASSACHUSETTS CHARITABLE EYE AND EAR INFIRMARY. 69th Annual Report, 1894.

7. PRESBYTERIAN EYE, EAR, AND THROAT CHARITY HOSPITAL OF BALTIMORE. 17th Annual Report, 1894.

8. NEW YORK EYE AND EAR INFIRMARY. 74th Annual Report, 1894.

9. MANHATTAN EYE AND EAR HOSPITAL. 25th Annual Report, 1894.

10. BROOKLYN EYE AND EAR HOSPITAL. 26th Annual Report, 1894.

11. OPHTHALMIC HOSPITAL OF CINCINNATI. Report for 1894, Drs. Ayres, Sattler, and Goode.

12. NEW YORK OPHTHALMIC AND AURAL INSTITUTE. 25th Annual Report, including a history of the institution, 1894. Dr. H. Knapp, executive surgeon.

A short guide to the study of the normal anatomy of the eye cannot well be analyzed. But the text and the drawings of the book by ROCHON-DUVIGNEAUD (1) are a valuable contribution. The chapter on the ciliary region is particularly noteworthy.

SULZER.

The Incas and Mamas put artificial eyes of gold into their mummies, and artificial eyes are also found in Egyptian mummies, according to PANSIER (2). The sclera was composed of a white mass, the iris of obsidian. In Rome there were makers of artificial eyes for statues. It is impossible to fix the epoch at which artificial eyes were first worn by living individuals, but it was probably when Egyptian culture was at its height. Ambroise Paré in 1579 gave the first description of artificial eyes, which were then made of enamelated gold and the color was similar to that of the human eye. SULZER.

During the year 1894 these were treated at the Massachusetts Eye and Ear Infirmary (6) 14,260 new eye patients. The customary good statistical report of the cataract operations shows 138 extractions. Of these 27 were simple, 84 with iridectomy, and 27 with a button-hole in the iris. In four there was no perception of light after the operation ; in one fingers were counted at 10 *cm*; the remainder (except 3 who died) had vision ranging from 0.02 to 1. The deaths were in no way connected with the operations. BURNETT.

During the year 1894 those were treated at the Presbyterian Eye, Ear, and Throat Hospital of Baltimore (7) 8521 eye cases. The cataract extractions were: 74 with iridectomy, and 53 simple; strabismus operations, 72; iridectomies, 62; enucleations, 38. BURNETT.

This report of the New York Eye and Ear Infirmary for 1894 (8) gives a description, with illustrations, of the fine new edifice which has been completed and occupied since the last annual report. The number of new eye patients during 1894 was 16,125. There were 86 simple extractions and 21 combined extractions. BURNETT.

The twenty-fifth annual report of the Manhattan Eye and Ear Hospital (9) gives a résumé of its work for its first quarter of a century's existence. During the last twelve months, 11,201 new eye patients were treated and 1470 operations performed, including the minor ones. There were 106 simple cataract extractions and 2 with an iridectomy. The total number of the patients treated since the opening of the hospital (including ear, throat, and nervous) is 177,898. A number of interesting statistics are included, such as the number of patients under 9 years of age, the religion of the applicants, the number refused treatment, number of prescriptions dispensed, number of days' board both pay and free, and the nativity of the patients. BURNETT.

The number of new eye patients at the Brooklyn Eye and Ear Hospital (10) for the year 1894 was 9018. There were 20 extractions of cataract and 6 without iridectomy. BURNETT.

In the Ophthalmic Hospital of Cincinnati (11) there were treated during the year 1894, 2304 eye patients. There were performed 267 major and 195 minor operations. There were 26 simple extractions and 13 combined extractions, 22 enucleations, and 51 iridectomies. BURNETT.

The number of new eye patients treated at the New York Ophthalmic and Aural Institute (12) for 1894 was 7500. There were 688 operations on the eye, of which 163 were extractions of cataract; of the extractions 128 were simple extractions, of which 118 were good, 8 moderate, and 1 failure. To this report is added a brief sketch of the history of the Institute since its foundation twenty-five years ago. In this are noted not only the gradual growth and development of the hospital as a clinical and educational institution, but an account of its recent enlargement and great improvement in its facilities for work. The total number of new patients treated at the Institute during the twenty-five years was (including ear and throat) 157,471, and the total number of operations 15,017. BURNETT.

The 9th and 10th portions of Helmholtz's book, which has been carried on since his death by Prof. Koenig, contain the chapter on ocular movements, the monocular field of vision, the direction of vision, and the perception of depth.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

13. VENNEMAN. The organization of blood clots in the eye. *Acad. de Méd.*, 1895.

VENNEMAN (13) made experiments in regard to the organization of blood clots in the eye, in order to try to obtain better conditions for the wearing of an artificial eye. When in a rabbit's eye or a human eye, the blood remains in the exenterated ball, its organization forms a solid stump. After incomplete exenteration in rabbits, and after supraretinal hemorrhage in man, Venneman has seen the pigmented cells of the retina proliferate and wander into the clot. They mingle here with the young cells which arise from the proliferation of the fixed connective-tissue cells. It is undecided whether or not the pigment cells of ectodermic origin take part in the formation of the connective tissue which replaces the clot. SULZER.

III.—INSTRUMENTS AND REMEDIES.

14. BARABASCHEW. On formaldehyd. *Wjest. Opht.*, 1895, No. 2.

15. HOLTH. On the proper receptacles for the preservation of the yellow salve. *Norsk. Mag. f. Lagevid.*, Nos. 1, 5, 13, Christiania, 1895.

16. SEGAL. On some objective methods of determining refraction (sciascopy, vasoscopy, the method of Schmidt-Rimpler and that of Fick). *Wjest. Opht.*, 1895, No. 1.

17. SULZER. Note on the construction of contact glasses. *Soc. d'opht. de Paris*, Jan. 8, 1895.

18. GROENOUW. Ephedrin-homatropine solution, a mydriatic

of very transient effect. *Deutsche med. Wochenschr.*, 1895, No. 10, p. 161.

19. Bull, C. S. The question of the efficacy of subconjunctival injections of mercuric bichloride in ophthalmic therapeutics. *New York Med. Journ.*, Jan. 19, 1895.

20. Davis, A. E. Double movable mires for Javal-Schiötz ophthalmometer. *New York Med. Journ.*, Feb. 16, 1895.

Barabaschew (14) gives first an abstract of the literature of the antiseptic properties and medicinal uses of formaldehyd, which he has used for a year, regarding it as the best antiseptic in ophthalmic practice. $\frac{1}{4000}$—$\frac{1}{2000}$ solutions are active, do not irritate, and can be used continuously. Stronger solutions produce irritation and should be used only after cocainization. When the eyes are tired, the instillation of a few drops of a weak solution is refreshing. In cases of trachoma the cartarrhal symptoms pass off and the granulations become smaller. The use of a $\frac{1}{1000}$ solution with cocaine cuts shorts the course of blennorrhœa, and acts favorably in cases of infectious keratitis. Atropine, cocaine, and such drugs, in a $\frac{1}{4000}$ solution, keep for months ; and for the disinfection of instruments, formaldehyd is very serviceable.

Hirschmann.

Holth (15) found that exposure to the air did not injure the yellow salve, no matter what its strength, but that exposure to light was the cause of its decomposition and turning gray, and he therefore advises that it be kept in porcelain jars with opaque covers.

Schiötz.

The comparison of a number of methods of determining errors of refraction leads Segal (16) to the conclusion that sciascopy ranks in accuracy near the subjective method and the determination in the erect image, but, possessing the added advantage of simplicity, is one of the best methods. The methods of Schmidt-Rimpler and Fick are useless in practice.

Hirschmann.

Sulzer (17) discusses in detail the construction of contact glasses. The blown glasses are optically imperfect, and are liable to break and injure the eye, but are lighter and have smoother edges than the ground glasses. The best glasses have been ground first and then the edges made smooth by a heating process.

Groenouw (18) in 100 cases employed a solution of ephedrine hydrochlor. 1.00, homatr. hydrochlor. 0.01, and aq. dest. 10.00, and found that in eight minutes there was noticeable dilatation of the pupil, which reached its maximum in half an hour and disappeared in 4–6 hours. The reaction to light is abolished, but the accommodation is not affected, and there is no irritation. The solution given above proved to be the most satisfactory.

BULL (19) has given subconjunctival injection of mercuric bichloride a trial in those classes of cases for which it has been recommended, and finds that it is undoubtedly useful, but the great pain which almost always follows its use is a great barrier to its employment. The only cases in which the sublimate injections seemed to exert any positive effect in allaying the severity of the symptoms and shortening the duration of the process were those of scleritis and acute irido-choroiditis of the non-syphilitic type.

BURNETT.

DAVIS (20) has made a modification of the instrument of Javal whereby the two mires can be moved simultaneously. For low degrees of astigmatism, 1 to 4 D, and where the angle alpha is small he finds this is of no great advantage, but in higher degrees and with a large angle alpha, there appears to be some superiority in exactness over the original method of moving only one mire. The method of adapting the instruments now in use to the new modification is described. BURNETT.

IV.—ANATOMY.

21. GABRIÉLIDÈS. Studies on the embryology and comparative anatomy of the angle of the anterior chamber in the chick and in man. The dilatator pupillæ. *Arch. d'opht.*, xv., No. 3, p. 176 (with illustrations).

GABRIÉLIDÈS (21) studied the development of the region of the iris angle in chick and in human embryos and adults, and draws the following conclusions : 1. The ligamentum pectinatum remains in the chick but disappears in the human fœtus after the seventh month, thus widening the anterior chamber. 2. The posterior wall of Schlemm's canal is formed, both in man and in the chick, by the continuation of the corneal endothelium, which lines the interior of the anterior chamber completely. 3. The ciliary muscle in the chick consists of an anterior and a posterior portion, while in man the ciliary muscle corresponds to the posterior portion only. In the chick the ciliary muscle begins at Schlemm's canal, in man at the iris angle. The so-called Henle's layer of the iris does not exist in the chick, but in its place is a layer of striped muscular fibres, which extend, gradually becoming thinner, from the connective tissue of the ciliary processes to the circular muscle. The same layer of muscular fibres exists in man, and is nothing else than the dilatator pupillæ. v. MITTELSTÄDT.

V.—PHYSIOLOGY.

22. KATZ. On fatigue of the eyes and the determination of its degree. (The effect on the eye of different sorts of illumination.) *Wratsch*, Nos. 4 and 5, 1895.

23. TCHIRIEW. A new entoptic phenomenon. *Acad. des Sciences*, Nov. 26, 1894.

24. STETTLER. Has the thickness of the lines of the test types an influence on the determination of the acuteness of vision. *Beiträge z. Augenh.*, xviii.

25. MADDOX. Simple optical notes. *Ophth. Review*, xiv., p. 67.

26. LAWFORD. The visual tests for railway servants and marines. *Brit. Med. Journ.*, March, 1895, p. 641.

27. PRENTICE. The sphero-toric lens in astigmatic aphakia. *Ophth. Record*, Jan., 1895.

28. PRENTICE. A problem in bifocal lenses solved by the prism-diopter. *Annals of Ophth. and Otol.*, Jan., 1895.

KATZ (22) believes that in reading the fatigue of the retina (illumination), the fatigue of convergence (reading with adducting prisms), the fatigue of accommodation (reading with glasses), and the fatigue of the conjunctiva are indicated by frequent winking of the lids which increases with the fatigue. For registering the frequency of the blinking he used Marey's myograph. The author suggests this method for the comparison of different sorts of illumination.

On awaking in the morning TCHIRIEW (23) sees projected on the faintly-lighted white ceiling of his room, a network of squares, each corresponding to a retinal image of about 1 *mm* square. He offers no explanation of the phenomenon. SULZER.

STETTLER (24) made a number of experiments with pot-hooks of equal height and breadth, but with varying thickness of line, and found that the latter affected the acuteness of vision but slightly.

Maddox (25) points out that the focal length of a concave lens can be found by measuring the focal length of each surface by reflection, remembering that this is four times the focal length by refraction. He suggests the "catoptre" as a reflective unit which would therefore be equal to four dioptres by refraction. When an attempt is made to neutralize strong lenses by contact, it is found that the neutralization is never complete, owing to the distance apart of the surfaces not in contact; their effect can be represented by a meniscus lens produced by the intersection of the completed spheres of which the free surfaces form part.

In prisms, a line joining the image by internal reflection to the true image is absolutely parallel to the base-apex line. While if a cylinder be not cut quite parallel to the axis, the linear image by internal reflection will not appear to pass through the flame.

In speaking of cylinders the author uses as approximate indications the terms, crown-slant, chin-slant, brow-slant, etc. Maddox uses the expression, "aberration of the eye" to denote the want of coincidence between the axis of vision and the axis of the globe, and speaks of high and low aberration, normal and negative aberration. When a patient fixes an ophthalmoscopic mirror which throws light into the eye, the line uniting the centre of the mirror with the centre of curvature of the cornea, which is called "the line of the corneal image," lies between the visual axis and the axis of fixation. If the term "aberration" be used as a general term which supposes the visual axis and fixation axis to be practically one, then the line of the corneal image affords an index of the amount of aberration. Werner.

Lawford (26) gives instances of rejection for defective color vision, and hypermetropia respectively, in candidates for promotion in the service. Werner.

Prentice (27) shows in this paper the principle of the toric lens which has a surface with two unequal curvatures at right angles to each other, and points out its practical applicability in making glasses for aphakial eyes with considerable astigmatism, whereby the lenses are not only rendered thinner and more nearly periscopic, but have much less of those internal reflections which cause the "glare" so frequently complained of by the wearers of lenses with a strong convex curve. Burnett.

Prentice (28) points out in this paper why it is that in certain cases bifocal glasses are unsatisfactory, especially where there is an astigmatism. This is due to the vertical prismatic action brought about by the "wafer" which is cemented on the lower segment of the larger lens. The key to the solution of the difficulty is found in the law "that a lens decentred one centimetre will produce as many prism-diopters as the lens has diopters of refraction." Therefore, the lens from which the segmental wafer is to be cut must have a definite diameter for every combination, and for this reason lenses having diameters of at least 64 *mm* should be ground from which these segmental wafers can be cut.

BURNETT.

Sections VI.–XI. Reviewed by PROF. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

29. HOOR. Further contributions to school myopia. *Wiener med. Wochenschr.*, 1895, No. 18.

30. BOCK. A contribution to the statistics of myopia. *Das oesterreichische Sanitalswesen*, 1895, No. 4.

31. PERGENS. The correction of myopia by aphakia. *Zehender's klin. Montsbl.*, xxxiii., p. 42.

32. VOSSIUS. The operative treatment of myopia, with remarks on cataract operation. *Beiträge z. Augenh.*, xviii., p. 48.

33. SCHANZ. On the increase in acuteness of vision by the operative treatment of high myopia. *Graefe's Archiv*, xli., 1, p. 109.

34. DE WECKER and MASSELON. On the advantages of convex glasses for distant vision in myopes. *Ann. d'ocul.*, cxiii., p. 83.

35. CHAUVEL. Acuteness of vision of astigmatic patients. *Rec. d'opht.*, 1895, No. 1, p. 10.

Among 96 myopic recruits between the ages of twenty-one and twenty-three, HOOR (29) found only 36 who had been long at school, and in the others there could not be traced the influence of any deleterious near work.

Among 8306 eye patients BOCK (30) found 673 myopes, 110 with progressive myopia, and 124 with poor vision. There were many more male myopes than female. Of 11 with a myopia of 20 D or more there were only 5 who were not peasants.

In 11 cases PERGENS (31) made discission of the lens of the left eye, without subsequent detachment of the retina or other complication. The decrease in refraction varied between 14 and

18 D, and the acuteness of vision was increased in almost all cases.

Vossius (32) operated on 9 eyes of patients between the ages of seven and twenty-three, with myopia varying from 10 to 30 D. The acuteness of vision was improved in all.

When a convex glass is held before a myopic eye at such distance that the focus of the glass falls in the punctum remotum of the eye, the combined system is adapted for parallel rays. The image is inverted, and for a given convex glass the magnification is proportional to the degree of the myopia, while for a given degree of myopia the magnification is greater the weaker the convex glass. De Wecker and Masselon (34) have in this way given an acuteness of distant vision to myopes double that obtained by concave glasses. They recommend convex glasses of about 6 D.

VII.—LIDS.

36. Bayer, F. Demonstration of a case of malignant pustule of the lids. *Corr. Bl. d. Ver. Deutscher Aerzte in Reichenberg*, 95, No. 3.

37. Braquehaye and Sourdille. On calcified epithelioma of the lids. *Arch. d'opht.*, xv., 2, p. 65.

38. Ahlström. On ectropion operations. *Eira*, 1895, No. 2.

39. Katz. Abnormal association of motion of the upper lid and iris with movements of the eyeball. *Wratsch*, 94, No. 46.

40. Hansell. A case of formation of a new lid by transplantation of skin without pedicle (Wolfe's method). *Med. News*, Mar. 9, 1895.

Bayer's (36) case of anthrax carbuncle of the lid was in a boy of ten, whose father had been employed with fresh skins of animals, whence the infection was supposed to have arisen. The case was cured after operation and the use of warm applications

Herrnheiser.

In the case reported by Braquehaye and Sourdille (37) a tumor the size of a bean, hard but movable on the bone, was found at the upper orbital margin in a man of forty. It was extirpated and showed an enveloping membrane from which many tracts of connective tissue passed inward containing in their meshes cells similar to pavement epithelial cells with chalky granules scattered through their protoplasm—a calcified epithelioma. This variety of tumor has been long recognized though it is fairly rare, and is found at every age and usually in the orbital region. Notwithstanding its cancerous structure the tumor is benign and grows slowly or may remain stationary. The diagnosis can only be made with the microscope. Histologically two forms may be differentiated, that in which the connective-tissue tracts are calcified and show Haversian canals, and that in which only the cells are calcified. The authors believe the theory of its congenital origin to be the most probable. There is probably an invagination of epidermis into the subcutaneous tissue either congenital or traumatic. The treatment is extirpation. Scraping is not sufficient. V. MITTELSLÄDT.

In ectropion operations, according to AHLSTRÖM (38), one must endeavor to bring the anterior margin of the lid to a level with the posterior, for only in these circumstances will the subtarsal muscle be able to functionate. The author's method of operation is as follows: The lid, fixed by a Jaeger clamp, is split behind the cilia in the usual fashion. The sutures are threaded each on two needles, one of which is passed through the conjunctival flap from behind forward at a distance of 1 *mm* from the flap margin. The needle emerges in the wound and is then passed upward under the skin into the region of the orbital margin. The second needle on the same thread, without piercing the conjunctival flap, is passed into the wound and up under the skin in the same manner as the other and is brought out 5 *mm* from it. The two ends are then tied fast over a small drainage tube. In case the lid is very much relaxed a triangular flap of skin is removed with its base at the margin of the anterior flap of the lid. SCHIÖTZ.

In the case of KATZ (39) the left palpebral fissure stood higher and was somewhat wider than the right. In looking down, the left upper lid did not follow the motion of the eye which had normal mobility in this direction. The mobility of the ball was limited inwards and upwards, and the upper lid did not follow the motion completely. The left pupil was moderately dilated (4 *mm*) and did not react to light or convergence but contracted in looking down and dilated again in looking up. Accommodation almost totally absent. R OD blurred. R V = 0.3; L V = 0.8. Beginning tabes. Improvement under the use of salicylate of soda.

The author supposed that there were changes in the nucleus of the left oculo-motor nerve and the association fibres. The nuclei were more or less atrophic, the connection between the depressors of the eye and the elevators of the lid destroyed, the accommodation abolished, and the reflex arc for the light reaction interrupted. A new connection had been formed between the inferior rectus and the sphincter of the pupil, which had possibly existed before the disease began, in the form of connecting fibres between the nuclei of the nerves or of a connecting branch between their peripheric portions. HIRSCHMANN.

HANSELL (40) reports a case of transplantation without a pedicle and its condition nine months after the operation. The graft had, at that time, shrunken to $\frac{2}{3}$ its original area, but the skin, though whiter than the surrounding parts, appeared healthy.

BURNETT.

VIII.—LACHRYMAL APPARATUS.

41. ADLER. Mumps of the lachrymal gland. *Wiener med. Presse*, 1895, No. 1.

42. BAYER, F. The modern treatment of dacryocystitis. *Corr. Bl. d. Ver. D. A. in Reichenberg etc.*, 1895, No. 3.

43. SCHÄFFER. A case of sarcoma of the lachrymal gland. *Inaug. Dissert.* Giessen, 1895.

In a boy of eighteen, with mumps, ADLER (41) noticed swelling of both lachrymal glands as well as the parotid, sublingual, buccal, and maxillary glands.

BAYER (42) opposes the passage of probes as the chief treatment of chronic dacryocystitis, and recommends the following procedure: The lower canaliculus is split, astringents are instilled, and the patient is told to keep the contents of the sac diligently pressed out. If this treatment is insufficient a probe should be passed after cocainization, in order to determine whether a stricture is present. If there is no stricture stronger astringents are injected with an Anel's syringe. When the bone is affected, Bayer has had good results from the use of injections of iodoform glycerin (1:10). HERRNHEISER.

SCHÄFFER (43) reports a tumor half as large as a walnut originating in the lachrymal gland, which was removed with preservation of the ball, and proved to be a small, round-celled sarcoma. There was no recurrence.

IX.—MUSCLES AND NERVES.

44. LANDOLT. Clinical observations on the surgical treatment of strabismus. *Arch. d'opht.*, xv., 3, p. 144.

45. KUNN. Congenital defects in the motility of the eyes. *Beiträge z. Augenheilk.*, xix., p. 1.

46. SCHILER. Congenital paralysis of ocular muscles inherited through three generations. *Med. Corr. Bl. d. Würtemb. A. V.*, 1895, No. 4.

47. HEINTZ. On a case of bilateral paralysis of the abducens after diphtheria without other ocular disturbance. *Centralbl. f. prakt. Augenheilk.*, 1895, p. 33.

48. MARTIN. Treatment of certain paralyses of the external rectus. *Annal. d'ocul.*, cxiii., p. 53.

49. GINSBERG. Herpes zoster frontalis with neuro-paralytic keratitis and oculo-motor paresis. *Centralbl. f. prakt. Augenheilk.*, 1895, p. 133.

50. SABRAZÈS. Does an hysterical nystagmus exist? *Semaine méd.*, Sept. 26, 1894.

51. HANSELL. The etiology and treatment of internal strabismus. *Journ. Amer. Med. Assoc.*, Feb. 16, 1895.

LANDOLT (44) offers two new clinical histories in proof of his views on the advantage of advancements over tenotomies. A teacher, aged twenty-two, had a divergent strabismus of her right eye of 27°, dating from childhood. Vision nearly normal in each eye. Both interni were advanced, and a careful tenotomy of the right external rectus done. Ten days later there was homonymous diplopia, and the tenotomized external rectus was advanced. The result was good, the abduction being normal, the adduction two metre angles greater, and binocular fixation constant. In a man of thirty-two the interni had been tenotomized several times apparently for esophoria. There remained convergence and great limitation of abduction. Advancement of one externus and stereoscopic practice restored the muscular balance and relieved the subjective symptoms.

SCHILER (46) observed in a man of sixty-three complete immobility of the eyeballs, and in a son aged twenty-eight rotatory nystagmus and deficient power of convergence. The patient could not look to the side; there was moderate ptosis, $V = \frac{1}{10}$; accommodation normal.

In four cases of unilateral paralysis of the external rectus of unknown origin, MARTIN (48) obtained a cure by injecting bichloride subcutaneously. Some of these cases were recent, and some of as long as three months' standing. Eight to fourteen injections were sufficient. SULZER.

Charcot and his pupils believed that there was always an anatomical change underlying nystagmus, either in the eye or the nervous system. SABRAZÈS (50) observed in a girl of seventeen a twitching of the right hand and a lateral nystagmus that he regarded as hysterical. When the lids were closed the nystagmus ceased at once. Both symptoms had come on suddenly a few days before in a fit of anger. A year before, in convalescence from typhoid, a twitching of the right hand had been noticed lasting two weeks. There was also somnambulism and concentric contraction of the visual fields, and by exclusion the author came to the conclusion that the symptoms were of hysterical nature. SULZER.

The general conclusions reached by HANSELL (51), after a long review of the facts and theories of squint and its attendant phenomena, is that the amblyopia of squint is congenital and not acquired, and is not improved by tenotomy when of any considerable degree. It is always present in monocular squint, but can be removed when the other hitherto good eye is lost or seriously injured in its visual power. In monocular squint vertical equilibrium (which is often disturbed) must be restored by tenotomy, while in the alternating form the simple section of the interni is sufficient.
BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

52. WALTER. Bilateral gumma of the orbit ; with autopsy. *Zehender's klin. Monatsbl.*, xxxiii., p. 8.

53. VAN DUYSE and BRIBOSIA. Enophthalmus with voluntary intermittent exophthalmus. *Arch. d'opht.*, xv., 3, p. 159.

54. MERZ. A case of orbital phlegmon after empyema of the antrum of Highmore. *Zehender's klin. Monatsbl.*, xxxiii., p. 55.

55. PANAS. Empyema of the maxillary sinus complicated with orbital osteo periostitis with perforation of the roof ; abscess of the frontal lobe and atrophy of the optic nerve ; death. *Arch. d ophth.*, xv., 3, p. 129.

56. KUHNT. On the inflammatory affections of the frontal sinus and their consequences. Bergmann, Wiesbaden, 1895.

57. CHIBRET. Salicylate of soda in exophthalmic goitre. *Rev. génér. d'opht.*, 1895, No. 1.

In a three-year-old foundling WALTER (52) saw a bilateral exophthalmus develop to such a degree that both eyes were lost from corneal ulceration. The child died, and at the autopsy it was found that there had been a general development of syphilitic gummata throughout the body and also in the orbits.

In a miner, aged twenty-nine, after a severe contusion in the right orbital region, VAN DUYSE and BRIBOSIA (53) saw develop an enophthalmus which changed to an exophthalmus when the body was bent over or the jugular vein compressed. There was no vascular change in the region of the disc, no scars or varicose veins in the region of the orbit, and no signs of pulsating exophthalmus. In the opinion of the authors this condition was due to atrophy of the fatty tissue of the orbit from neuro-trophic disturbances and a varicose dilatation of the orbital veins.

v. MITTELSTÄDT.

Following caries of two molar teeth MERZ (54) saw an empyema of the antrum develop, followed by an orbital phlegmon. Either the floor of the orbit was perforated or the nose was purulently infiltrated and then the orbit tertiarily affected. After extraction of the teeth, removal of the pus from the orbit, and washing out of the orbit, antrum, and nose, the case was cured.

A clerk, aged thirty-one, with empyema of the antrum of dental origin, suddenly lost the sight of the right eye as PANAS (55) reports, and at the same time there developed symptoms of orbital phlegmon. The disc was pale, the arteries small, the veins dilated. After opening and draining the antrum and orbit the vision improved. Then a meningitis developed suddenly and the patient died. At the autopsy the right frontal sinus was found filled with pus, the antrum communicating with the orbit where the periosteum was detached on all sides by the pus, while the orbital contents otherwise were not involved. There was no thrombophlebitis. In the region of the optic foramen there had been perforation into the cranial cavity, and corresponding to the spot of perforation was a small cerebral abscess. There were signs of inflammation of the optic nerve, particularly about the optic foramen.

v. MITTELSTÄDT.

Having observed 24 cases of frontal sinus disease, KUHNT (56), taking into account also the literature, gives a detailed description of this affection, its clinical history, etiology, pathology, complications on the part of the orbit, eye, and brain, and its therapy. Fourteen of his own cases were operated upon.

In four cases of characteristic and fully developed Graves's disease, Chibret (57) obtained good results by using large doses of salicylate of soda, on the supposition that most of these patients belong to families with a tendency to arthritic affections.

Sulzer.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

58. Silex. Statistics of ophthalmia neonatorum. *Zeitschr. f. Geburtshilfe u. Gynäkol.*, xxxi., 1.

59. Parinaud and Doyen. On the treatment of purulent ophthalmia. *Annal. d'ocul.*, cxiii., p. 78.

60. Cohn. The prevention of ophthalmia neonatorum. *Centralbl. f. prakt. Augenheilk.*, 1895, Nos. 107 and 136.

61. Fromaget. The treatment of purulent ophthalmia with formal. *Annal. d'ocul.*, cxiii., p. 87.

62. Walter. Gonorrhoic ophthalmia. *Wiener klin. Wochenschr.*, 98, Nos. 10 and 12.

63. Eliasberg. On pseudo-membranous conjunctivitis. *Wjestnik Ophth.*, 95, No. 2, p. 194.

64. Gayet. A trial of sero-therapy in diphtheritic conjunctivitis.

65. Hoppe. A case of diphtheria of the eye and throat treated with antitoxin. *Deutsche med. Wochenschr.*, 95, No. 12.

66. Schmidt-Rimpler. Trachoma and follicular conjunctivitis; their treatment by means of the roller forceps. *Berliner klin. Wochenschr.*, 95, No. 1.

67. Syronajatschnikow. The operative treatment of trachoma by expression. *Wojeno Med. Journ.*, 1895, No. 1.

68. Tschirikow. The treatment of trachoma by expression with Knapp's roller forceps. *Russkaja Med.*, 95, Nos. 7-9.

69. Rabinowitsch. The operative treatment of xerophthalmus. *Wjest. Ophth.*, 95, No. 2, p. 200.

70. Wagenmann. A case of local variolous inflammation of the conjunctiva. *Graefe's Archiv*, xli., 1, p. 172.

71. Puech and Fromaget. Serous subconjunctival cyst of dermoid origin. *Ann. d' ocul.*, cxiii., p. 268.

72. Rombolotti. A clinical and anatomical study of serous or simple cysts of the conjunctiva. *Ann. d'ocul.*, cxiii., p. 161.

73. Rogmann. A case of carcinoma of the limbus conjunctivæ in a child aged fourteen. *Ann. d'ocul.*, cxiii., p. 178.

74. Galtier. Subconjunctival osteoma. *Ibid.*, p. 186.

75. BORTHEN. On pemphigus of the conjunctiva. *Zehender's klin. Monatsbl.*, xxxiii., p. 158.

76. STÖWER. Bleeding from the conjunctiva of the lid. *Deutsche med. Wochenschr.*, 95, No. 6.

77. FRIEDENWALD. Ante-partum ophthalmia neonatorum (intra-uterine ophthalmia). *Med. News*, March 9, 1895.

SILEX (58) finds that notwithstanding the knowledge of Credé's method, blennorrhœa of the new-born still occurs, and he would advise making the prophylactic measure obligatory.

COHN (60) also believes in making Credé's procedure obligatory.

FROMAGET (61), in cases of purulent keratitis, advises irrigations with a 1:2000 solution of formal alone, or in severer cases combined with the use of nitrate of silver. SULZER.

ELIASBERG (63) describes two cases of pseudo-membranous conjunctivitis. He does not advise using nitrate of silver until the discharge becomes purulent. HIRSCHMANN.

In an infant aged sixteen months with unmistakable diphtheria of the conjunctiva, GAYET (64) made injections of antitoxin without the slightest result. V. MITTELSTÄDT.

In HOPPE'S (65) case a child aged two had diphtheria of the pharynx and eyes. The cornea was cloudy. After injections of antitoxin the diphtheritic symptoms disappeared and the cornea gradually cleared up.

According to SCHMIDT–RIMPLER (66) trachoma and follicular conjunctivitis are two distinct affections. He recommends Knapp's roller forceps in the treatment of trachoma.

SYRONAJATSCHNIKOW (67) gives the results of his experience during two years in the treatment of 128 trachoma patients. Expression led to recovery in from two weeks to a year in 50 per cent. of cases, in 36 per cent. of which no scars remained.

Expression is contra-indicated: (1.) when there is marked conjunctivitis; (2.) when there is papillary hypertrophy; and (3.) when there is marked cicatrization of the conjunctiva. The author used the forceps of Dohnberg and those of Bellarminoff.

HIRSCHMANN.

TSCHIRIKOW (68) recommends the treatment with Knapp's roller forceps. In 93 cases there was complete recovery. There were no recurrences or severe complications. The average duration of the treatment was 4–5 weeks. HIRSCHMANN.

RABINOWITSCH (69) reports that Pagenstecher had done the operation proposed by Rudin for twenty-five years with varying success. In one case Rabinowitsch had a good result that was only temporary. In three months the palpebral fissure was widened again and the xerosis returned. HIRSCHMANN.

WAGENMANN (70) reports the case of a sister of charity in whose conjunctiva several variola pustules developed. These were caused by direct transference of variolous matter by the water in which a small-pox patient was bathing, flying into the eye. The affection was cured completely without leaving any traces.

ROMBOLOTTI (72) observed the formation of a serous cyst of the conjunctiva in the lower retrotarsal fold of a boy aged twelve. Microscopic examination showed it to be the greatly dilated duct of a Krause gland. The interior of the cyst was lined with flattened epithelium. At most points there was a double epithelial layer, at others a layer even thicker. The stratum next the connective tissue consisted of cuboidal cells with large nuclei, the other strata of flattened cells. SULZER.

ROGMANN'S (73) case of epithelioma of the limbus conjunctivæ in a boy of fourteen is a new instance of the benign nature of these tumors. Simple extirpation brought about a permanent recovery. Rogmann had a similar favorable result from the operation six years ago of an extensive epithelioma in a man of sixty-five. SULZER.

In literature there are but three observations of subconjunctival osteoma. In all these cases the tumor was located in the upper retrotarsal fold. GALTIER (74) removed from the same location a bony, hard tumor, measuring 7 x 10 *mm.* It consisted of true bone included in a connective-tissue sheath. No cartilage was found. The child, six months before the tumor made its appearance, fell, striking the supraorbital region, and Galtier believes that the tumor represented the further development of a splinter of bone chipped off from the supraorbital process. SULZER.

In a woman of seventy-four, with pemphigus of the neck, BORTHEN (75) saw symblepharon develop in both eyes, and opacity of the cornea in the right.

STÖWER (76) saw severe bleeding from the conjunctiva of the upper lid in an infant of seven months. A new growth with a granular surface was found under the lid, which was taken to be a granuloma developing from a chalazion that had broken through the conjunctiva. It was excised and the wound scraped. There was but slight bleeding at the operation.

FRIEDENWALD (77) gives in this paper the abstracts of the histories of 18 cases of intra-uterine ophthalmia, and adds two new cases, one from his own practice, in which the conjunctivitis was in existence when the child was born. The mother had chancrous and venereal warts. The child was delivered three hours after the breaking of the membranes. BURNETT.

78. BOCH. Experimental studies on staphylococcus ulcer of the cornea and its therapy. *Graefe's Archiv*, xli., 1, p. 56.

79. ZIMMERMANN. A case of keratitis parenchymatosa tuberculosa. *Ibid.*, p. 215.

80. HALTENHOFF. The treatment of keratoconus. *Ann. d'ocul.*, cxiii., p. 60.

BOCH (78) produced ulcers of the cornea by inoculating with staphylococcus pyogenes aureus. He then made subconjunctival injections of bichloride. These had not the slighest therapeutic value, but rather increased the irritation of the eye.

Sections XII.-XXI. Reviewed by DR. SILEX.

XII.—IRIS.

81. BÜRSTENBINDER. On tuberculous iritis and parenchymatous keratitis. *Graefe's Archiv*, xiv., p. 85.

82. LAGRANGE. A case of primary tuberculosis of the ciliary body and iris. *Arch. d'opht.*, xv., No. 3, p. 170.

83. SILEX. Demonstration of two cases of ocular tuberculosis. *Berl. med. Gesellsch.*, Sitzung v. Feb. 20, 1895.

84. BAYER. Demonstration of a case of iris tuberculosis. *Correspondenzbl. d. A. in Reichenberg etc.*, 1895, No. 3.

Two forms of iris tuberculosis are well known—first, the usual progressive form with the development of nodules, and second, the so-called attenuated tuberculosis of Leber. BÜRSTENBINDER (81) calls attention to the fact that many cases of serous and plastic iritis are really of tuberculous nature, a view which Michel has taken in his text-book. A tuberculous child received a blow on the head, after which an iritis without nodules developed, which was supposed to be due to an embolic process. He reports also 3 cases of parenchymatous keratitis thought to be tuberculous.

LAGRANGE (82), in a child of seven, of healthy parents and with no other signs of scrofula or tuberculosis, saw a tuberculous tumor of the ciliary body which had broken through the sclera, causing an intercalary staphpyloma. v. MITTELSLÄDT.

The first patient presented by SILEX (83) was a girl of thirteen with general tuberculosis, in whom tuberculosis of the left iris had developed seven weeks before. Since enucleation was not done the growth of the yellowish-red nodules could be observed and the clinical picture was exactly that of inoculation tuberculosis in rabbits. Silex stated that in man this form of tuberculosis is never cured, nor is it influenced by treatment.

The second patient, a boy of six, had suffered for two years with extensive tuberculous ulcers of the right eyelids, which grew worse until the child was sent to the country, when recovery soon took place.

XIII.—CHOROID.

85. ZIMMERMANN. A case of suppurative irido-choroiditis with complete recovery and restoration of vision. *Zehender's klin. Monatsbl.*, xxxiii., p. 45.

86. VALUDE. Septic irido-choroiditis following uterine hemorrhage. *Ann. d'ocul.*, cxiii., p. 38.

ZIMMERMANN'S (85) case was one of gonorrhoic purulent irido-choroiditis. Injections of bichloride, which were used together with salicylate of soda, atropine, and hot applications, were supposed to have been chiefly instrumental in bringing about recovery.

XIV.—GLAUCOMA.

87. WALTER. The etiology and therapy of glaucoma. *Zehender's klin. Monatsbl.*, xxx., p. 1.

88. SIMI. Glaucoma. *Boll. d'ocul.*, xvi., 12, p. 93.

89. RADSWITZKI. An acute attack of glaucoma following the use of cocaine. *Wjest. Ophth.*, 1895, Jan.–Feb.

90. ADAMÜCK. Two cases of malignant glaucoma. *Arch. f. Augenheilk.*, xxx., 2–3, p. 242.

91. LODATO. Iridectomy in congenital hydrophthalmus. *Arch. di ottalm.*, ii., 6, p. 187.

92. WILLETTS, JOSEPH E. The halo or rainbow symptom in glaucoma. *Annals of Ophth. and Otol.*, Jan., 1895.

It has been stated that scopolamine is less likely to do injury to eyes predisposed to glaucoma than atropine or hyoscine.

Walter (87), however, has seen two cases of glaucoma due to its employment, both cured by eserine. The drugs have no specific effect in causing glaucoma, but do it only by dilating the pupil.

Simi (88) saw a severe attack of glaucoma cured by massage and the use of eserine.

Radswitzki (89). In an eye on which iridectomy had been done, instillation of a 4 % solution of cocaine twice brought on glaucoma, which was relieved by sclerotomy.

Adamück's (90) patients had each lost an eye from glaucoma, and in each the second eye became affected, and, in spite of iridectomy, went on to blindness. From this he concludes that in such cases it is best to perform iridectomy on the eye first blind in order to see its effect. If the course of healing is complicated in the first, the second should be treated with myotics or by sclerotomy or paracentesis of the cornea.

Lodato (91), after considerable experience, declares iridectomy to be the best means of preventing the further development of hydrophthalmus, and the operation should be done as early as possible. Dantone.

Willetts (92) considers that the phenomenon of the halo in glaucoma is due to stasis and œdema of the cornea, and not to a non-eliminated excretion in the blood. He draws this conclusion from experiments he has made on pigs' eyes. Burnett.

XV.—SYMPATHETIC OPHTHALMIA.

93. Jocqs. On a case of sympathetic keratitis. *Soc. d'opht. de Paris*, March, 1895, and *Ann. d'ocul.*, cxviii., p. 202.

Under the title sympathetic keratitis Jocqs (93) gives the following clinical history: A woman of fifty-eight who had lost the left eye at the age of thirteen from an unknown cause, had pain and irritation in the phthisical eye and at the same time a painful infiltration of the cornea in the other eye, which improved at once after removal of the phthisical eye.

XVI.—LENS.

94. Bjerrum. The antiseptic measures to be taken in cataract operations. *Med. Aarsskrift*, p. 151, Copenhagen, 1895.

95. Schuleck. On a new method of removing cataracts. *Ungarische Beiträge zur Augenheilk.*, Vienna, 1895.

96. SCHULECK. A concave flap extraction with preservation of a round pupil. (3 % of failures). *Ibid.*

97. TENNANT. A simplification of the cataract operation. Dr. Trousseau's procedure. *Thèse de Paris*, 1894.

98. CHIBRET. A new method of clearing out cortical masses after cataract extraction. *Soc. d'opht de Paris*, Feb. 5; and *Ann. d'ocul.*, cxiii., p. 120.

99. ZENKER. A thousand cataract operations. Report from the practice of Duke Carl of Bavaria. Wiesbaden, 1895.

100. JUHÁSZ. The cataract operations in Prof. Schuleck's clinic in the years 1876–79. *Ungarische Beiträge z. Augenheilk.*, Vienna, 1895.

101. ISSEKUTZ. Experiences in cataract extraction in the years 1880–90. From Prof. Schuleck's clinic. *Ibid.*

102. GASPARRINI. Hemorrhage in both eyes following extraction spontaneously recovered from. *Ann. di ottalm.*, xxiii., 3–4., p. 270.

103. MERZ. Iridocyclitis after extraction, secondary glaucoma, sympathetic affection of the second eye and secondary glaucoma in it. *Zehender's klin. Monatsbl.*, xxxiii., p. 50.

104. MARTIN. The word "astigmia." Astigmia after extraction.

105. HIRSCHBERG. On lamellar cataract. *Centralbl. f. Augenheilk.*, xiv., p. 75.

106. DEMICHERI. False lenticonus. *Ann. d'ocul.*, cxiii., p. 93.

107. STORY. A series of one hundred cataract extractions. *Brit. Med. Journ.*, Jan., 1895, p. 198.

108. SYM, W. G. A case of lenticonus posterior. *Ophth. Rev.*, xiv., p. 76.

BJERRUM (94) recommends the following procedures preparatory to cataract extraction. 1. Treatment of any existing lachrymal trouble with probes, injections, or extirpation of the sac. 2. Careful cleansing of the skin about the eye the day before, and the day of the operation, with warm 1:1000 bichloride solution. 3. Washing the lid margins and conjunctiva with 1:5000 bichloride solution the day before, and again immediately before the operation. He advises against closing the canaliculi, epilating normal cilia, or applying caustic solutions to the conjunctiva and then bandaging the eye, which latter should never be done as a preparatory measure. SCHIÖTZ.

SCHULECK (95) has a peculiar method of pressing out the lens. The capsule is opened with Förster's forceps.

TENNANT (97) has taken for the subject of his dissertation the frequently described operation of Trousseau. The linear knife is the only instrument employed. The statement of Tennant that the danger of infection is proportional to the number of instruments used is subject to criticism. SULZER.

CHIBRET (98) has arranged a syringe with a double canula which injects a quantity of fluid into the anterior chamber and simultaneously aspirates an equal quantity. The instrument is used for cleansing the anterior chamber after extraction, and has made a secondary operation unnecessary in thirty consecutive cases. SULZER.

In his monograph ZENKER (99) reports on 1000 extractions done by Duke Carl Theodor within four years, grouping them into series of 100, on each of which critical remarks are made. The chief operation is the extraction with iridectomy and the section in the limbus. The capsule is opened with forceps. The combined operation is preferred to the simple, as prolapse of the iris does not occur, and the after operations required are fewer. "How the eye looks is a matter of indifference to the patient, providing he sees." The patient usually remains eighteen days in the hospital, three days on liquid food, and five days with both eyes bandaged. Great stress is laid on practising the patient in moving the eye as required in the operation. If there is any difficulty from that cause, chloroform is used. The results were good in 95.2 %, fair in 3.2 %, and poor in 1.6 %; loss of the eye from suppuration occurring in 0.9 %. After-operations were done in 139 cases, mostly with Knapp's discission needle, one eye being lost from suppuration after the operation.

GASPARRINI (102) reports the case of a cataract patient in whom extraction in both eyes was followed by repeated hemorrhages in the eyes, which finally ceased, leaving relatively good vision. DANTONE.

MARTIN (104) proposes to replace the word astigmatism by astigmia. The Greek word *στίγμα, ατος* means prick and not point, as Whewell, the coiner of the word astigmatism, believed. Point, in a mathematical sense as well as in the meaning luminous point is expressed in Greek by the word *στιγμή, ῆς*. Astigmia is therefore not only shorter and more convenient, but also more correct etymologically.

The rule that the astigmia after cataract extraction decreases for three months and then remains stationary does not always hold good. Martin saw an astigmia of 3.25 D decrease 1.75 D three years after the operation. In another case the astigmia increased for a time, and later became less. SULZER.

HIRSCHBERG (105) has operated on 168 eyes for lamellar cataract, 63 of the total 99 patients being males. The treatment is threefold: (1) to do nothing; (2) to do iridectomy; and (3) to remove the lens and preserve a round pupil. He has made 114 iridectomies, 50 discissions, 1 division of iris and capsule, 8 partial extractions, and 9 flap extractions. The theoretical observations on the optical disadvantages of iridectomy in such eyes are valuable, and the more so since they coincide with the views of Schweigger, in whose clinic iridectomy for lamellar cataract has been done but six or eight times in fifteen years.

By the expression "false lenticonus," DEMICHERI (106) means the condition of difference in refraction between the cortex and the nucleus of the lens in beginning cortical cataract, which at a superficial examination may be taken for lenticonus. The changes in the refraction of the lens often cause a marked diminution of vision when the opacity is still scarcely recognizable. The myopia coming on in some cases of senile cataract is of the same nature. When there is a considerable difference in refractive index between the peripheric and central portions of the lens, it may happen that the nucleus becomes visible as such in transmitted light. Different methods of examination show in these cases that the rays near the axis are more strongly refracted than those which pass through the periphery of the lens. The observation of Purkinje's images shows that these refractive anomalies, which are identical with those observed in cases of keratoconus and lenticonus, do not depend upon a change of form in the refracting surfaces. This view is confirmed by the fact that Demicheri has been able to see the reflected image from the nucleus in one of his patients in whom the peripheric and central portions of the lens differed markedly in refractive index.

STORY'S (107) cases included 42 simple extractions, and 12 after preliminary iridectomy. He had not made up his mind as to the superiority of either method. In the simple operation he had 12 per cent. of prolapses, but on the whole vision was slightly better than in the combined operation. Infection occurred, with loss of vision, twice. WERNER.

SYM'S (108) patient, a woman, æt. fifty-two, had high myopia in both eyes, with choroidal changes in the affected eye, which had vision = fingers at 7 *m* with — 15 D. With retinoscopy the pupillary area presented a clear outer zone, then a dull ring enclosing a bright central area in which the vessels appeared to move with greater velocity. An image of a flame on the posterior lental surface appeared much smaller than in the other eye, and its movement was less. No other abnormality in the eye.

WERNER.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

109. DEUTSCHMANN. On a new method of treating detachment of the retina. *Beiträge zur Augenheilk.*, 1895.

110. CASPAR. On the etiology of retinal striations. *Arch. f. Augenheilk.*, xxx., 2–3, p. 122.

111. KUHNIG. On the etiology of amaurosis. A further contribution to the statistics of blindness from the university eye clinic in Greifswald. *Inaug. Dissert.*, Greifswald, 1894.

112. GROCZ. A case of total blindness caused by an anthelmintic. *K. Aerzte-Verein zu Budapest.*

113. DE BONO. Quinine amaurosis and amblyopia. *Arch. di ottalm.*, ii., 5–6, p. 146.

114. EWETZKI. Bilateral recurrent amaurosis with consecutive temporal hemianopsia. *Medicinskoge obosrenije.*, xliii., 2, 1895.

115. BJERRUM. On intoxication amblyopia. *Med. Aarsskrift*, p. 194, Copenhagen, 1895.

116. SCHULECK. Erythropsia "blood spots" on the snow and seeing red in dim light. *Ungarische Beiträge z. Augenheilk.*, Vienna, 1895.

117. BAYER. Displacement in the visual field with scotoma scintillans. *Twenty-sixth Vers. d. südwest deutschen psych. Vereins*, Karlsruhe, 1897.

118. LAQUEUR. On a case of embolism of the central artery of the retina with preservation of the temporal region of the retina, with remarks on the centripetal pupillary fibres. *Arch. f. Augenheilk.*, xxx., 2–3, p. 75.

119. SILEX. The albuminuric retinitis of pregnancy. *Berl. klin. Wochenschr.*, No. 18, 1895.

120. TROUSSEAU. Recurrent hemorrhage into the retina. *La clinique opht.*, Jan., 1895.

121. BARRETT. Damage to vision caused by watching an eclipse. *Ophth. Review*, xiv., p. 80.

122. Reber. Six instances of color-blind women occurring in two generations of one family. *Med. News*, Jan. 26, 1895.

123. Smith, H. E. Peculiar perversion of the color perception. *Med. Record*, March 9, 1895.

124. Goode. Embolism of the inferior branch of the central retinal artery. *Annals of Ophth. and Otol.*, Jan., 1895.

In his views on the pathogenesis of detachment of the retina, Deutschmann (109) takes Leber's standpoint, and in his treatment his aim is to divide any bands attaching the shrinking vitreous to the retina, and permit the latter to rest again upon the choroid. This is accomplished by an incision which opens the subretinal space, passes through any folds of the retina, and then passes anteriorly. In some cases the point of a Paquelin cautery was passed into the subretinal space. When the result was slight the operation was repeated. In 1 case he obtained a good result in this way, but in 3 others there was a relapse, so the combination method was given up, and he returned to the simple cutting of the vitreous, which he did in 7 patients with good result.

Most cases were of myopic detachment, all the retinas were floating, and a spontaneous perforation was found in no case. The intraocular tension was normal in all.

In a second series of cases the procedure had only a transient effect, and in these the retina was not floating, they being the cases in which Nordenson believes the vitreous to be only detached from the retina in a slight extent, and causing a gradual detachment of the retina, the fluid collecting beneath it. A case of this sort, with an exudation of serous fluid in the vitreous, running an unfavorable course after incision of the vitreous, led the author to the conclusion that recovery might be obtained under the following conditions: 1. The free fluid in the ball must be evacuated. 2. The preretinal space must be filled with a fluid which after the escape of the other fluid would press the retina against the choroid. 3. A mild irritation must lead to attachment of the retina to the choroid, but must be aseptic and must not injure the elements of the retina.

This he hoped to accomplish by injecting fresh vitreous from a living animal. The vitreous was rubbed up with some drops of physiological salt solution, and injected into an eye that had perception of light to one side only. Four weeks later, $V = \frac{17}{200}$, and it remained the same a year after.

Many other remarkable results are reported, and it is only to be hoped that others may obtain good results with his method, and that experience will not prove his method to be worthless like the others that have been enthusiastically recommended.

CASPAR (110) regards the so-called retinal striations as residua of cured detachments of the retina, developing from organized subretinal fibrinous masses that are replaced by connective tissue from the choroid growing into them, and pigmentary changes taking place.

GROCZ (112) patient took 8 grammes of extr. filicis maris and 8 grammes of extr. punicæ granati. Two days later he was completely blind with mydriasis and normal fundus. Ten days later the disc began to grow pale. The optic nerve atrophy that occurs in these cases is supposed to be peripheric. The extr. filicis is particularly toxic when it is given with ol. ricini.

DE BONO (113) observed three cases of quinine poisoning and describes the functional disturbances of vision. Experiments on dogs showed that subcutaneous injections of 0.25 quin. mur. for each kilometre of body weight produced no symptoms, 0.40 produced toxic symptoms, the vision often clearing up later but never becoming normal, 0.50 produced death. DANTONE.

EWETZKI'S (114) patient, a peasant aged nineteen, became suddenly blind after severe headache lasting a week. Perception of light and light reaction were completely wanting. Besides dilatation and tortuosity of the retinal veins there was nothing abnormal in the fundus. Two years previously he had become blind, and after using iodide of potassium for three weeks the blindness gave place to a bitemporal hemianopsia, which lasted four weeks and then passed off. The patient had acquired syphilis five years before, and now under specific treatment the blindness again gave place to bitemporal hemianopsia, and this in its turn to complete recovery of vision. Ewetzki localized the process in the lower surface of the chiasm, the gumma possibly springing from the sella turcica. HIRSCHMANN.

BJERRUM (115) treats briefly of the different forms of intoxication amblyopia. Under tobacco amblyopia he reports a case in which the affection was unilateral, and a second which in the beginning seemed to be a simple atrophy, but tests with small objects revealed a horizontally oval central scotoma. SCHIÖTZ.

SCHULECK (116) believes that erythropsia is the condition of regeneration or assimilation of the red-green visual substance after a destruction of it by photochemical excitation (ultra violet).

In the course of an attack of scotoma scintillans in himself, Bayer (117) noticed that portions of objects perceived in the lower right portion of the field were apparently transposed to the upper left portion of the field. He explains this transposition on the ground that the disturbance in the central visual spheres causes not only signs of irritation and defects but also a false blending of the excitations coming from the eye.

Laqueur's (118) patient was a girl of twenty with endocarditis and embolism of the central artery, but with $V = \frac{2}{3}$ in a very small irregularly rectangular field, which was supplied by a small branch given off from the main trunk behind the embolus. Sixteen such cases are on record. The fact that illumination of the blind portion of the retina produced prompt pupillary reaction seemed to the author to speak for the existence of so-called pupillary fibres which are more resistant than the visual fibres.

Silex (119) has made observations for seven years on a large material. He gives first a detailed description of the ophthalmoscopic picture and the pathological anatomy of albuminuric retinitis in general, and states that a differential diagnosis of the different varieties is not possible from the objective ophthalmoscopic condition alone. Hemorrhages seem to be more frequent in the retinitis occurring in pregnancy. He calls attention to a new vascular reflex which is yellow and broader than the normal arterial reflex, and which he believes to be due to a filling of the perivascular lymph spaces with serous fluid, and not to a hyalin degeneration of the vessel wall, since there may be complete restitution. The decrease in vision is slow and the affection appears particularly in primiparæ toward the end of pregnancy. The field of vision and color preception are not affected. The author believes that nephritis of pregnancy may pass over into chronic nephritis. The prognosis with retinitis from Bright's disease and genuine sclerosis of the kidney is very bad, and patients rarely live more than two years. The prognosis is better in acute nephritis, and is best in the retinitis of pregnancy. The acuteness of vision may often be restored, but usually secondary changes in the retina and choroid lead to marked decrease in vision. These secondary changes are the less frequent, the sooner after the beginning of the retinitis the pregnancy is terminated either naturally or artificially. When the urine of a pregnant woman contains albumen the eyes should be watched and with the appearance of retinitis abortion should be brought about to prevent loss of vision and guard against eclampsia.

Barrett's (121) patient watched an eclipse for a quarter of an hour with both eyes through four pieces of colored glass, two blue, one red, and one yellow. She did not see any after-image. A fortnight later print seemed hazy with this eye and the central letters of a word seemed to be missing. Vision and field normal except for small central scotoma 4 × 3 *mm* at a distance of 50 *cm.* Macropsia around the scotoma. Macula lutea a little larger and darker than in other eye. No change in three months. Werner.

Reber's (122) patient was a woman aged fifty, who stated she had been color-blind all her life and had two sons who were also similarly affected. (The confusion was of dark-green, dark-red, and brown. Rose and blue were confounded.) She stated that her three sisters also had impaired color-sense as well as an only brother. In their father's family, both of his brothers and his three sisters were color-blind. Burnett.

This most interesting case of Smith (123) occurred in a boy of eleven years who, with no previous illness, noticed dragging of the feet and hysterical symptoms. Three days later there was an inco-ordination of gait, tendency forward, and the child complained that white surfaces appeared brown and objects looked smaller and distorted. There was constant occipito-frontal headache, formication in arms and fingers, backache and pain during mastication; no nausea, vomiting, convulsions, or loss of consciousness. The motility was unimpaired, hand-grip stronger in left, no muscular twitchings or contractures; knee-jerk and ankle-clonus abolished. Hearing good. Vision $\frac{4}{20}$ in right, $\frac{6}{20}$ in left. No strabismus or conjugate deviation of the eyes, and ocular motions good; pupils equal and of normal size, and contract to light convergence and Wernicke's test. Fields contracted. Accommodation good. Selects colors correctly as to shade, but each appears of its complementary color—red looks like green and *vice versa*, and blue like yellow and *vice versa;* white looks like brown. No other color tests described. There is a monocular diplopia of each eye, which, however, is not accurately described. There was a diffuse neuro-retinitis of both eyes. Pulse was 64; respiration, 16 to 18; temperature, normal; taste and smell normal. The case was treated by five-grain doses of iodide of potassium three times a day, and in four weeks all the symptoms had disappeared. Smith makes an elaborate analysis of the symptoms, but is unable to give a solution of the phenomena except by locating the seat of lesion (probably a blood clot) in the left optic thalamus with pressure effects on the internal capsule. The perversion of the color sense—assuming that it is not hysterical—is not easy to account for. BURNETT.

In GOODE's (124) case there was an absence of the upper half of the visual field, and on examining the fundus carefully he saw a bulbous distention of the artery a short distance below the edge of the optic disc. There was mitral regurgitation and the patient had had rheumatism. Some months later the condition was unchanged. BURNETT.

XIX.—OPTIC NERVE.

125. PEPPMÜLLER. On the prophylactic and therapeutic value of resection of the optic nerve. *Inaug. Dissert*, Halle, 1895.

126. WAGENMANN. On a modification of resection of the optic nerve to guard against sympathetic ophthalmia. *Græfe's Archiv*, xiv., p. 180.

127. SCHEIDEMANN. A case of gummous new-formation at the point of entrance of the optic nerve. *Ibid.*, p. 156.

128. SULZER. An optic neuritis consecutive to ozæna. *Ann. d'ocul.*, cxiii., p. 1.

129. NAMMACK, CHAS. E. Syphiloma of optic chiasm producing bilateral temporal hemianopsia. *Med. Record*, Feb. 9, 1895.

PEPPMÜLLER (125) gives a sketch of the development of the theories of the causation of sympathetic ophthalmia, and of the experimental pathological investigations that have been made. His own work consists of the study of the resected portions of six optic nerves in which no micro-organisms or signs of inflammation were found. Of thirty-one reported cases of resection, four eyes were enucleated later for recurrent cyclitis in from one to three months. The second eye was not affected in a single case, but since sympathetic inflammation has been known to occur notwithstanding resection, he does not regard resection as the prophylactic equivalent of enucleation. He believes that it should be done, however, when enucleation is refused.

According to Gifford's and Deutschmann's investigations excision of a portion of the optic nerve does not necessarily make conduction through the nerve impossible. The latter found that both ends of the nerve were united later by a strand of connective tissue which communicated with the intervaginal space of both ends of the nerve, and clinically sympathetic ophthalmia has been seen to occur after resection. In order to make it a surer protection WAGENMANN (126) in two cases cauterized the cut ends of the nerve, studies on rabbits having shown him that union was thus prevented.

While the optic nerve is often secondarily involved in cases of syphilis, its primary involvement is rare. Gummatous formation behind the ball, marked unilateral choked disc, and interstitial neuritis have been seen. An intrabulbar gumma of the nerve, such as SCHEIDEMANN (127) observed in a man of thirty-two about ten months after infection, has not been previously reported. On the disc was a prominent broad-based, round, circumscribed nodule of light- or yellowish-gray color. The circumpapillary portion of the retina was involved in the swelling, the veins were dilated, and the arteries thin or even invisible, and there were striped hemorrhages. $V = \frac{1}{200}$. Under energetic treatment the symptoms mostly disappeared and vision increased to $\frac{15}{100}$. The choroid was not involved. The process probably started in the walls of the arteries, and later involved the veins ; during the stoppage of the former the ischæmia came on, and the lack of blood caused the venous extravasations.

SULZER (128) has observed several cases of mild optic neuritis in cases of ozæna. The acuteness of vision was increased by treatment of the nose. A certain correspondence of the subjective and objective symptoms of the different cases suggested the question whether there is not an etiological connection between essential ozæna and a certain form of optic neuritis.

NAMMACK (129) reports a case of a man thirty-four years of age who had an initial lesion in October, 1889. Six weeks later he had a paralysis of the right side for which he was treated by protoiodide of mercury, and later by iodide of potassium for four years. At the end of that time he was seen on account of headache. $V = \frac{20}{20}$ in either eye ; knee-jerks exaggerated ; ankle-clonus ; pupils normal. A typical temporal hemianopsia was found. He was given inunctions of blue ointment, and iodide of potassium internally. Improvement soon set in, and in a few months the fields were normal. BURNETT.

XX.—INJURIES, FOREIGN BODIES, PARASITES.

130. BADAL. Wounds of the eye by shot. *Soc. d'opht. et de laryng. de Bordeaux*, 1894, and *Annal. d'ocul.*, cxiii., p. 43.

131. Fromaget. A case of tetanus consecutive to a penetrating wound of the eye. *Ibid.*, p. 68.

132. Trousseau. A case of traumatic amblyopia. *Ibid.*, p. 119.

133. Goldschmidt. On removal of iron splinters from the interior of the eye with the electro-magnet. *Deutsche med. Wochenschr.*, Nos. 3 and 4, 1895.

134. Goldzieher. A case in which a splinter of copper remained in the retina ten years, with remarks on impregnation of the retina with copper (chalcosis retinæ). *Centralbl. f. Augenheilk.*, xix., p. 1.

135. Randolph. A suggestion as to the treatment of penetrating wounds of the ciliary region and lens. *N. Y. Med. Jour.*, Feb. 23, 1895.

Badal (130) has repeatedly enucleated eyes injured by shot without finding the foreign body in the ball. The shot lay in the orbit where it caused no disturbance. He therefore advises against enucleation in such cases. Sulzer.

In December, 1893, a girl was struck in the eye with a music roll. For two weeks there was inflammatory symptoms and later a progressive diminution of vision. In July, 1874, Trousseau (132) found complete amaurosis, a large pupil, and anæsthesia of the upper lid. In the upper retrotarsal fold was a bit of rolled-up macerated paper. After the removal of this foreign body there was complete recovery within two weeks. Sulzer.

Goldschmidt (313) reports three cases of extraction of iron splinters from the eye by means of the electro-magnet, in one case preserving normal vision. The author fully coincides with Hirschberg's views, and even goes so far as to say that the frequency of introduction of the magnet need not deter one from further attempts, and in the case in which he had the most favorable result the magnet was introduced eight times. The author thinks that oculists should advise the use of protecting glasses by metal workers.

In Goldzieher's (134) case the retina to the temporal side of the disc and in its anterior layers showed numerous orange, yellow, or red shining spots communicating with one another by means of linear processes. In the centre of this region lay a bit of gun-cap. $V = \frac{20}{30}$. In the vitreous there were some membranous opacities which may increase and later cause detachment of the retina, so that the eye is still in danger. The spots and lines were supposed to be due to oxide of copper.

The great dangers attending penetrating wounds of the ciliary region can be obviated, in great measure, in RANDOLPH'S (135) opinion, by a removal of the lens, and especially if this body participates in the injury. By doing this you not only avoid the evils of increased intraocular pressure, but with the removal of the lens you also get rid of at least a part of the septic material which has been enclosed in the eye. This should be done as soon as possible and under strictly aseptic conditions. He relates the history of three cases in which this mode of procedure was successfully put in practice. BURNETT.

XXI.—OCULAR DISTURBANCES IN GENERAL AFFECTIONS.

136. HERTEL. The relation of acromegaly to ocular affections. *Graefe's Archiv*, xiv., p. 187.

137. GALEZOWSKI. On hereditary ocular syphilis. *Soc. de dermat. et de syphilol. de Paris*, 1894, Nov. 15.

138. SILEX. The syphilitic affections of the eye. *Dermatol. Zeitschrift.*, 1895, vol. ii., part 2.

139. ADAMÜCK. On ocular affections following typhoid processes. *Petersburger med. Wochenschr.*, 1894, Nos. 38 and 39.

140. BERGER. Disturbances of sensibility of the eyeball and its adnexa in tabes. *Sem. Med.*, Nov. 21, 1894.

141. ARPAD KRIZ. An unusual case of severe puerperal pyæmia with many embolic abscesses but with a favorable termination. *Casopis lékarno ceskyct.*, 1894.

142. EVERSBUSCH. Treatment of the affections of the eye coming on with infectious diseases. (Reprint from the *Handbuch der speciellen Therapie inneren Krankheiten*, Fischer, Jan., 1895.

143. GOTTBERG. Loss of sight from a shot with suicidal intent. *Arch. f. Augenheilk.*, xxx., 2 and 3, p. 122.

144. SALTINI. Contribution to the study of some metastatic changes in the eye. *Ressegna di Scienze Mediche*, Modena, 1894.

145. HAHN. Pathological examination of Lissauer's case of psychical blindness. (*Cf. Arch. f. Psychiat.*, vol. xxi.) *Allgem. Zeitschr. f. Psych.*, v., p. 1006.

146. MÜLLER, FR. A contribution to the knowledge of psychical blindness. *Arch. f. Psych.*, xxiv., 3.

147. PICK. On the local diagnostic significance of visual disturbances in brain affections. *Prager med. Wochenschr.*, 1 and 2, 1895.

148. LUMBROSO and LEVI. Contribution to the electric reaction of the eye. *Boll. d'ocul*, xvi., 10, p. 94.

149. DE SANCTIS. New researches and new considerations on the visual field of the insane. *Riv. Speriment. di Trenatria et di Med. Legale*, xiv., 3–4.

150. NATHANSON. Ocular affections dependent upon general diseases of the organism. St. Petersburg, 1895.

151. DE WECKER. Does reflex traumatic amblyopia exist? *Soc. d'opht. de Paris*, March 5, 1895; and *Ann. d'ocul.*, cxiii., p. 203.

152. LAGRANGE. On monocular hysterical diplopia. *Rec. d'opht.*, No. 1, 1895.

153. SILEX. Disfigurement from syphilis. *Berl. med. Gesellschaft.*, Jan. 9, 1895.

Among 174 cases of acromegaly, HERTEL (136) found 91 with ocular disturbances. Some affections like conjunctivitis, opacities of the vitreous, and refractive errors were of course merely accidental, others were in immediate relation with the disease. Among these latter were abnormal thickening of the lids, both of the tarsus and the skin, and marked prominence of the orbital margins, so that the balls appear to lie deep in the orbit. In another case there was exophthalmus. A third group of symptoms were pain in the eyes, supraorbital neuralgias, lachrymation, hyperplasia of the lachrymal gland, nystagmus, oculo-motor pareses, lack of the ability to converge. The abducens was never affected. The fourth series were symptoms on the part of the optic nerve, neuritis or pressure atrophy, caused by thickening of the bones or enlargement of the hypophysis cerebri. In 11 published cases there was temporal hemianopsia, and choked disc in 5.

GALEZOWSKI (137) considers all severe syphilitic affections of the eye as tertiary. Iodide of potassium is of little value, and enunctions of mercury should be used. SULZER.

Among 1138 cases of typhoid, ADAMÜCK (139) had 32 ocular affections. The choroid was most frequently affected. The disease begins mostly some weeks after the recovery from the fever.

BERGER (140) reports 5 new cases of sensory disturbances of the eye and its adnexa. The cornea, conjunctiva, and the skin of the eyelids often show a diminished touch and warmth sense as well as loss of localizing power. The presence or absence of these disturbances is independent of the existence of gray atrophy of the optic nerve. SULZER.

The case of ARPAD KRIZ'S (141) was one of puerperal septicæmia, with many metastases and panophthalmitis of the right eye, notwithstanding which the patient recovered completely in five months. HERRNHEISER.

The treatise by EVERSBUSCH (142), which is of interest even to the specialist, is arranged in chapters dealing with the various portions of the eye. All the infectious diseases that may affect the eye, the connection between the two, and the clinical histories are carefully sketched out and the therapy taken into consideration, without too much reference to history and to views that have been in the course of time discarded.

GOTTBERG (143) reports four cases of blindness from shot injuries, and gives a *résumé* of the literature. If the ball is only grazed by the bullet, there will be hemorrhage into the vitreous and into the coats of the eye, and sometimes tearing of the latter. Injury of the optic nerve is always followed by atrophy. It may be injured in or behind the orbit by the ball itself, or indirectly by splinters of bone, hemorrhage, or callus formation.

SALTINI (144) describes four cases of metastatic inflammation in the uveal tract, one after puerperal infection, two after cerebrospinal meningitis, and one after vaccination. DANTONE.

One of MÜLLER'S (146) two patients was examined *post mortem*. There had been choked disc, decrease in vision, right hemianopsia, and psychical blindness. Objects were not recognized by sight alone, but only when they affected some other sense organ. At the autopsy a psammoma was found springing from the dura of the base of the left occipital lobe, with softening of the neighboring medullary layer.

PICK (147) gives a clear and comprehensive exposition of the localizing significance of visual disturbances in brain diseases.

HERRNHEISER.

LUMBROSO and LEVI (148) report their investigations on the excitability of the eyes to the electric current as regards light, color, and visual field in various nervous affections. DANTONE.

DE SANCTIS (149) observed anomalies of the visual field in 15 out of 30 insane patients. The contractions exceeded 15°. Excessive contractions or hemianopsia were found in none. Often the contraction was not constant, and the author thought it to be analogous to the variations in the visual field in normal individuals in whom slight contractions are found when the individual is tired, excited, or inattentive. DANTONE.

Lagrange (152) gives us a complete and well-worked-up monograph on monocular diplopia in the hysterical. He was led to this study by the observation of a case which in its symptoms differed from the majority, and which could not be explained in an optical way. A girl of twelve, after severe headache, sensitiveness of the left eye, and sleeplessness, suddenly developed marked and permanent diplopia of the right eye. This eye was normal and emmetropic, but vision was reduced to one fifth, and there was the characteristic hysterical dyschromatopsia. The conjunctiva of the ball was completely anæsthetic. The false image stood higher than the true one, and the distance between them was 4–5 *cm*, no matter what the distance of the object.

Hysterical monocular diplopia is of two varieties; in the majority of cases it is of optic origin. Irregular refraction, mostly due to irregular spasm of accommodation, causes two or more images to be thrown upon the retina. Besides this optic or peripheric hysterical monocular diplopia, which is mostly a polyopia, there is a central hysterical diplopia, probably due to a functional disturbance in the visual centres.

Central hysterical diplopia was described and explained by Duchesne de Boulogne, in 1864, and before him by Fallot. Of late it has been disregarded for peripheric hysterical polyopia. To Lagrange is due the credit of comparing the two affections and differentiating them. Sulzer.

Silex (153) presented a girl of twenty-four, in whom congenital syphilis had caused frightful disfigurement of the face and head. The patient had not been ill up to her twelfth year, when suppuration of the glands and ulcers in the face appeared, for which Bergmann ordered iodide of potassium five years ago. The cicatrization of the ulcers brought about the disfigurement. The septum of the nose was broken down and the eyelids shortened, causing lagophthalmus, and ulceration of the corneæ had rendered the girl practically blind. Upper lids were successfully made by grafting flaps from the arm at several sittings, and with this protection the corneæ became clearer and vision improved.

BOOK REVIEWS.

Perimetry. By Dr. Ole Bull of Christiania. Octavo, pp. 218. Friedr. Cohen, Bonn, 1895.

Bull, whose earlier studies on color and light sense are well known, has now given us in book form the practical results of his perimetric examinations on a material consisting of 12,000 eye patients. His own examinations only have been utilized, the observations of others being but briefly mentioned, and while this is in some respects commendable it nevertheless renders the book less valuable as a work of reference, since in a material so small, comparatively speaking, some anomalies of the visual field will not be met with and many will not be seen in sufficient number to enable one to clearly define the type of field characteristic of a disease and to note its variations.

As test-objects he has used the four colors, made of equal light intensity, and gray instead of the customary white. This is an advance on the methods employed by previous writers on perimetry, since a gray composed of $\frac{9}{10}$ white and $\frac{1}{10}$ black will be seen in as large a field as white in the normal eye but it will indicate marked contraction of the field in many pathological cases in which for white the field may be complete. For this reason Graefe long ago suggested reducing the general illumination when testing with white in doubtful cases.

Bull begins his examination with determining, as Förster did at first, the location and extent of the blind spot, since the field is more symmetrical reckoning from it than from the point of fixation. The limits of the field are usually larger than the normal in hyperopes and smaller in myopes. In cases of congenital red-green blindness or of complete color blindness, there is no contraction of the field for gray.

The main portion of the book is divided into two sections, treating respectively of the visual field in affections of the coats of the eyeball, and of the visual field in affections of the visual apparatus behind the eyeball. In myopia of high degree there are concentric contraction of the field, often enlargement of the blind spot, and at times small scotomata near the point of fixation, even when no macular changes can be seen with the ophthalmoscope. In syphilitic retinitis there may be found three types of scotoma, often with but slight visible change in the fundus and with good central vision. They are: 1st, a wing-shaped scotoma, single or double, beginning at the blind spot; 2d, a crescentic scotoma usually concentric with the disc; and 3d, an irregular ring scotoma including the blind spot. The determination of these scotomata and of the changes they undergo is of great value in prognosis. Since these scotomata are often relative in part and have ill-defined limits, they cannot well be made out with white test-objects, and the author therefore uses colored test-objects for this purpose and often finds the scotoma for red much larger than that for blue. But just in these retinal affections there is great confusion of color perception, red being mistaken for yellow and blue for green, and the reviewer has found it more advantageous to use black points of different size or gray patches of different intensity, the larger spots and darker patches often revealing no scotoma at all or a very small one, and the smaller points and paler patches revealing very extensive scotomata. In ordinary disseminate choroiditis the functional disturbance is frequently greater or less than the ophthalmoscopic picture would lead one to believe. With a gray test-object a concentric contraction can usually be made out, and with small colored objects the scotomata corresponding to the individual patches can be found. In retinitis pigmentosa there is usually concentric, but often irregular, contraction of the visual field, much more marked for gray test-objects than for white. In other cases there are ring-scotomata without much concentric contraction, or there are defects in the intermediate zone which do not form a complete ring. A point of some importance that might have been mentioned here is that defects limited to the intermediate zones of the field are found only with fundus diseases. In some cases of retinitis pigmentosa the defect in the intermediate zone may be in a measure symmetrical in the two eyes, and thus lead to a suspicion of disease behind the eye; but lesions in the chiasm or behind it, while they may cause defects in the central or in the peripheric zone only, never cause defects in the intermediate zone alone. In cases of glaucoma at all advanced, the author finds with gray test-objects a contraction above and below as well as inward, and in his opinion the most common form of field is an oval, with the least contraction outward.

While the ophthalmoscope is of greater importance in diagnosing diseases of the eyeball proper, the perimeter is of far greater importance in diagnosing diseases of the visual apparatus behind the eye. In cases of choked disc there is usually no change in the field, but in the higher grades of hyperæmia there is moderate concentric contraction. If the blind spot is enlarged and its margin not sharp there is also retinitis present. In optic-nerve atrophy there is no typical field, although the re-entrant angles are somewhat characteristic. Both eyes are usually affected, and a certain symmetry is often found in the field of the two eyes, particularly in cases having a spinal origin. Toxic amblyopia is treated at some length, and a possible distinction is made between hysterical and neurasthenic concentric contraction of the visual field, in that the limits of the field in hysteria seem to shift oftener in the course of the examination than those of neurasthenia.

Under hemianopsia are grouped binasal and monocular hemianopsia, which are never pure forms, and which many authors do not class as hemianopsia proper. The subject is treated under the three headings: bilateral, bitemporal, and binasal hemianopsia. It would certainly seem better to call the first variety homonymous simply, and reserve the expression bilateral hemianopsia for those cases, of which a number are on record, in which, for example, after a left homonymous hemianopsia a second lesion causes a right homonymous hemianopsia, leaving only a small central portion of the field free.

The book as a whole is the record of careful observation, forming a valuable contribution to the subject and a safe guide to the student. The determination of the visual field—that portion of the functional examination of the eye which is second in importance only to the determination of the acuteness of vision—has always been, and is to-day, too much neglected, and it is to be hoped that this book, which no one can read without profit, will be an incentive to more careful routine work in this direction.

W. A. H.

Prof. C. Schweigger's **Vorlesungen über den Gebrauch des Augenspiegels** (Manual of Ophthalmoscopy), rearranged and enlarged by Dr. RICH. GREEFF, Berlin. Octavo, 171 pages; 41 drawings in the text. Wiesbaden: J. F. Bergmann, 1895.

This treatise in its new form will be read with interest and profit by any student of ophthalmology. After a good introduction of fourteen pages on mirrors and lenses, the eye is considered as an optical instrument, and the discovery and construction of the ophthalmoscope and the ordinary methods of ophthalmoscopic examination are described. A short chapter is devoted to the ophthalmoscopic instruments, of which we transcribe the following passage, p. 39, below: "A. Roth has lately succeeded in constructing a mirror with an automatic combination of the lenses. The displacement of the line of combination, which hitherto had to be made with the hand, is done by the onward rotation of the disc itself, and Roth's instrument presents a progress in the construction of ophthalmoscopes. . . . An ophthalmoscope lately devised by Dr. Knauer, works in a similar way, and is commended by those who have used it."[1]

The special part treats of the examination of the refractive media and the normal background and its abnormities. Then a chapter on the ophthalmoscopic determination of the refraction of the eye is wedged in, in which the different old and new methods are described: that in the erect image, that in the inverted image with Schmidt-Rimpler's apparatus, that with the electric ophthalmoscope according to Schweigger, which is peculiar and particularly appropriate to measure high degrees of myopia. The image of a wire loop of an electric ophthalmoscope, formed in a tube by a strong lens of about 1″ focus, is looked at by patient and physician. When it appears clearest to the physician it indicates the inverted image of the patient's retina in the patient's far point. Its distance from the patient's iris is measured by an assistant, and represents the degree of the patient's myopia. Schweigger claims that by this method high degrees of M. can be far more accurately determined than by any other objective method. In skiascopy (shadow, or, better, illumination test), Schweigger prefers to render all eyes strongly myopic, and to read off the distance of the inverted image by a tape measure, attached to the ophthalmoscope, instead of finding the position of the point of reversal by lenses held before the patient's eye. An instrument of A. Roth is described, consisting of six colored lenses and a tape measure with six colored stripes, on which, without calculation, the refraction can be directly read off.

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[1] The two instruments are very neatly made in New York by E. B. Meyrowitz. They are double-disc ophthalmoscopes with a tilting mirror, accordng to Loring, have a superflous wealth of glasses—that of Knauer an uninterrupted series from 1. D to 39. D both + and — spherical. The two discs are moved by an ingenious device of transmission from one wheel to the other, but when we want to advance by 0.5 D, which near the zero point is quite important, our finger has to leave the milled edge of the disc and each time shove a 0.5 lens up the stem of the instrument, as in the older patterns of artery forceps. These instruments are less inconvenient than other double-disc ophthalmoscopes, but with regard to easy handling are still inferior to the single-disc instruments, and these can be made to carry a number of glasses sufficient for all purposes. There is in ophthalmoscopes the same superfluity of glasses as in microscopes. Four objectives, three dry and one oil immersion, suffice for all microscopic work, and from twenty-four to thirty lenses for all ophthalmoscopic work.

The last three chapters are devoted to the description of the ophthalmoscopic appearances of the diseases of the choroid, the retina, and the optic nerve. The descriptions are, perhaps, not so systematic and exhaustive as they might be ; some subjects are suppressed, others slighted ; for instance, the manifold primary and subsequent appearances of retinal hemorrhages, as well as the vascular changes, leave much to be desired ; the same may be said of the presentation of the affections of the optic nerve. The strong feature of the book is the constant reference of the ophthalmoscopic pictures to the pathologico-anatomical changes that produce them.

There are one or two mistakes in the treatise that should not be left unmentioned. The most striking is that on page 47, illustrated by a figure, to localize opacities, for which the point of rotation of the eye is made use of. This point is wrongly placed close behind the lens in the anterior part of the vitreous—*i. e.*, about 7.5 *mm* behind the cornea. The centre of rotation lies 1–2 *mm* behind the centre of the eyeball, namely, about 13.5 *mm* behind the cornea. It is an imaginary point and not available for ophthalmoscopic determinations. Opacities of the media, from the anterior surface of the cornea to the retina, can only be projected on two planes, the pupillary plane, and the plane of the centre of curvature of the cornea. With regard to the former, all opacities in the pupillary area keep their relative positions in all movements of the eyeball, remaining stationary in the red field of the pupil ; those situated in front of the pupil move with the cornea, those behind in an opposite direction through the red pupil.

The author's figure on p. 47 refers to the second plane, *O* is not the centre of rotation of the eye, but the centre of curvature of the cornea, which is evident by their erroneous explication of the appearance of the posterior polar cataract, p. 56, "which appears in the red background of the pupil as a dark patch close by the corneal reflex. It does not change its place during the movements of the eye, or, what is the same, it keeps its apparent position close by the point of rotation in all movements of the globe." Not only the explanation, but also the observation is wrong. The posterior polar cataract (not of very rare occurrence) coincides with the corneal reflex when the patient looks directly forward, but is noticed slightly above the corneal reflex when the patient looks up, and slightly below when the patient looks down. The reason of this is that the centre of curvature of the cornea (which on the observer's visual line always lies in the prolongation of the corneal reflex) is situated in almost all eyes about 0.5 *mm* behind the posterior pole of the lens.

Another mistake is on p. 101, where in the determination of astigmatism in the erect image it is said, that the weakest concave glass which makes a retinal vessel distinct shows the refraction in this meridian. It should read, "in the meridian at right angles," just as in the inverted image. One or two of such mistakes or oversights, do not essentially diminish the value of a book. They, as a small number of shortcomings in the description, are fully compensated by the lucid presentation of the bulk of the material. The author's familiarity with the subject and extensive personal experience manifest themselves on every page by a mathematical accuracy and conciseness of expression which delight the advanced student, though they may, at first, repulse the freshman. The chapter on the determination of the refractive state may serve as an example of this kind of description. The book can be heartily recommended to the student, who, not satisfied with the acquisition of a smattering of ophthalmoscopy, has the earnest desire to become proficient in one of the most elegant and useful departments of physical diagnostics. H. K..

Obituary.

I.—Henry Willard Williams, A.M., M.D., was born in Boston, December 11, 1821. He was educated at the Boston Latin School, but on account of ill-health was obliged to give up his preparation for college, and for about seven years he devoted himself to mercantile pursuits in a counting-room on Central Wharf and as Secretary of the Anti-Slavery Society. In 1844 he entered the Harvard Medical School, and from 1846 to 1848 inclusive, he studied medicine abroad, in Paris, Vienna, and Londan, devoting a considerable portion of his time to the diseases of the eye. On his return to Boston in 1849, he received his degree of M.D. from the Harvard Medical School, and during the cholera epidemic of that year he served as one of the Assistant Physicians to the Cholera Hospital. For two years he was one of the District Physicians to the Boston Dispensary, and was afterward its Ophthalmic Surgeon. In 1850 he was appointed Instructor in Theory and Practice of Medicine in the Boylston Medical School, which position he held for five years. From 1864 to 1891 he was Ophthalmic Surgeon to the Boston City Hospital, a period of twenty-seven years of active work, not only as attending surgeon, but also as a teacher, in utilizing the large clinical material for purposes of instruction. In 1866 he was made Lecturer on Ophthalmology in the Harvard Medical School, and in 1871 he was made Professor of Ophthalmology in this school, which chair he filled for twenty years—until 1891. He was President of the Massachusetts Medical Society from 1880 to 1882, and of the American Ophthalmological Society from 1868 to 1873. His first essay on the *Treatment of Iritis without Mercury*, published in 1856, led to a marked change in the treatment of this disease, in which thorough mercurialization had previously been considered as one of the first requisites. In 1862 he published a

Practical Guide to the Study of the Diseases of the Eye, which passed through several editions, and in 1881 was entirely re-written and appeared as his *Diseases of the Eye.* In 1865 he received the Boylston Prize for his essay on *Recent Improvements in Ophthalmic Science*, and he has been a frequent contributor to medical journals of articles on ophthalmic subjects. In 1868 he received the honorary degree of A.M. from Harvard University. He was a member of many societies, both medical and other, and retained his interest in all matters, and even continued his consulting practice, until a few weeks before his death, which occurred June 13, 1895, at the ege of seventy-three years.

II.—Dr. ALBRECHT NAGEL, Professor of Ophthalmology at the University of Tübingen, Würtemberg, died in the summer of 1895. He is chiefly known by the *Jahresbericht für Ophthalmologie*, which he founded about twenty-five years ago, and has conducted a number of years. He is the inventor of the *meter-lens*, which led to the introduction of the metrical system into practical ophthalmology both as applied to accommodation and refraction and to motility, the prism dioptry, etc. He has published a considerable number of original investigations, made partly by himself, partly by his pupils, in his *Mittheilungen aus der Ophthalmiatrischen Klinik in Tübingen*, 2 volumes, and in different periodicals.

III.—Dr. VIALET, of Paris, well-known by a recent excellent investigation on the localization of visual defects.

IV.—Dr. T. INOUYE, of Tokio, died July 10, 1895, from the consequences of a fall from a horse—complicated fracture of the femur and meningitis. He studied in Berlin and other cities, and was one of the chief promoters of scientific ophthalmology in Japan.

APPOINTMENTS AND HONORS.

Prof. SCHLEICH has been appointed to succeed the late Prof. Nagel, his teacher, in the chair of ophthalmology at the University of Tübingen, Würtemberg.

The Gräfe medal has been awarded to Prof. THEO. LEBER, of Heidelberg.

Prof. J. HIRSCHBERG, of Berlin, has been honored with the title of Geheimer Medicinalrath (Privy Medical Councillor).

Prof. WM. CZERMAK of Innsbruck has been appointed Professor of Ophthalmology in the German University of Prague.

Prof. WICHERKIEWICZ in Posen, Professor of Ophthalmology at Cracow, in place of Prof. Rydel.

Dr. MAX. SALZMANN, Lecturer (Privatdocent) on Ophthalmology at the University of Vienna.

Contents of the last number of the Arch. f. Aug. (Germ. Ed. of these ARCH.) Vol. XXXI., No. 2. Issued August, 1895.

1. JAESCHE, On Binocular Vision. (Four figures in the text.)

2. G. ADELSDORFF, On the Clinical Significance of Bitemporal Defects of Vision. (Twelve figures on two plates.)

3. Z. GUTMANN, Clinical Contributions to the Knowledge of the Tumors of the Eye. (Six figures in the text and a lithographic plate.)

4. L. BACH, On the Influence of Antiseptic Dressings on the Presence of Germs on the Edges of the Lids and in the Conjunctiva.

5. JUL. VON SIKLOSSY, On Blepharoplasty. (Nine figures in the text.)

Report on the Progress of Ophthalmology, 1st quarter of 1895. (Translation in the present number.)

INDEX OF SUBJECTS AND AUTHORS.

Abducens Nerve, Traumatic Paralysis of the, 243, 363
Advancement, Advantages of, over Tenotomy, 525
ALLING, Diplopia at the Periphery of the Field of Fixation, 365
Anatomy, Report on, 114, 268, 414, 518
"Annales d'Oculistique," English Edition of the, 143
Appointments, 140, 557

BAAS, Tuberculosis of the Lachrymal Gland, 76
BACH, L., Tuberculous Infection of the Eye, 43
BECKER, Microphthalmus with Orbito-Palpebral Cyst, 73
BLACK, A New Method of Tying the Sutures in Advancement of the Ocular Muscles, 375
Book Reviews, 139, 444, 549
BROSE, A Dermoid Tumor Involving the Cornea, with Remarks, 479
BRUGGER, Hyaline and Amyloid Degeneration, with Calcareous Degeneration and Ossification in an Ocular Muscle after an Injury, 65
BRUGGER and WEISS, Oxycephaly, and Disease of the Optic Nerve, 55
BURNHAM, Treatment of Syphilitic Affections of the Eye by Mercury, Potassium Iodide and Pilocarpine Combined, 406

Cataract, Glaucoma Following Discission of, Secondary, 433 ; 1,000 Cases of, Extraction of, 535 ; Spontaneous Resorption of, 510
Choroid, Double Coloboma of the, 202 ; Report on, 126, 286, 430, 532 ; Three Unusual Cases of Degeneration of the, 334 ; Tumors of the, 450
Ciliary Body, A Tumor of the, 161 ; Changes in the, Following Puncture of the Anterior Chamber, 81
Ciliary Region, Anatomy of the, 270
Cold, Use of Continuous, in Eye Diseases, 268
Color Centre, Non-Existence of a Cortical, 447
Conjunctiva, Cornea, Sclera, Anterior Chamber, Report on Diseases of the, 123, 280, 425, 528
Conjunctivitis, Diphtheritic, Treated with Antitoxine, 472
Cornea, Dermoid Tumor of the, 479
CUTLER, Congenital Night Blindness and Pigmentary Degeneration, 313 ; Three Unusual Cases of Retino-Choroidal Degeneration, 334

Diplopia in the Periphery of the Field, and the Diagnosis of Muscular Paralysis, 491
DODD, Diabetic Retinitis, 206
DUANE, Diplopia in the Periphery of the Field of Fixation, and its Bearing on the Diagnosis of Muscular

Paralysis, 491 ; The Parallax Test for Heterophoria, 258
DUANE and KNAPP, Permanent Scotoma and Revolving Hemianopsia by Looking at the Sun, 104
DUNN, A Case Illustrating the Bad Effects which the Establishment of Menstruation may have upon the Course of Interstitial Keratitis, 353 ; Congenital Monolateral Fistula of the Lachrymal Sac Situated above and Parallel with the Inferior Canaliculus, 251

Enucleation and Fatal Meningitis, 286
Eye Salves, Aseptic Value of, 265

Filiaria in Human Eye, 284
FINLAY, Alveolar Fibro-Sarcoma of the Optic Nerve, 224
Fixation, Diplopia of the Periphery of the Field of, 365
Fixation Spots, Two, in the Same Eye, with Diplopia and Divergent Strabismus, 246
Foreign Body, Death from a, Lodged in the Orbit, 425
Formaldehyd, 517
Formol, 413
FRIDENBERG, The Lens Star-Figure of Man and the Vertebrates, 154
Fundus Oculi, Anomalies of the Ophthalmoscopic Images of the, 192, 197

GIFFORD, An Orbicularis Pupillary Reaction, 402 ; Case of Hyaline Bodies in the Optic Nerve, 395
Glaucoma, Artificial Production of, and the Channels of the Eye, 169 ; Cases of Hereditary, 287 ; Report on, 127, 287, 431, 532
Glaucoma Simplex, Report of One Hundred and Seventy-Five Cases of, 378
GRADLE, The Palpebral Form of Spring Catarrh, 466
GREEFF, The Changes in the Ciliary Body Following Puncture of the Anterior Chamber, 81
GUTTMANN, A Case of Diphtheritic Conjuntivitis Presenting Unusual Complications, Treated with Antitoxine, 472

HANSEN GRUT, Strabismus Theories, 1
HARVEY, A Case of Anomalous Diplopia, Divergent Strabismus with Homonymous Diplopia, Two Fixation Spots in the Same Eye, 246
HECKEL, A Case of Successful Extraction of Steel from the Iris in which a Plastic Inflammation had been Established, Restoration of Vision, 482
Heterophoria, The Parallax Test for, 258
HOLDEN, On the Probable Hemorrhagic Origin of the Striated Affections of the Retina, 147 ; Observations on Cases of Hemichromatopsia, Indicating the Non-Existence of a Separate Cortical Color Centre, 447
HOLTH, Light—the Ointment of the Yellow Oxide of Mercury—and the Ointment Pots in Common Use, 486
Hyaline and Amyloid Degeneration in a Muscle after an Injury, 65

Injuries, Foreign Bodies, Parasites, Report on, 134, 294, 439, 543
INOUYE, Obituary Notice of, 556
Instruments and Remedies, Report on, 112, 266, 412, 515
Iris, Extraction of Steel from the, 482 ; Report on Diseases of the, 125, 285, 428, 530

JOHNSON, Observations on the Macula Lutea, 301

Keratitis, Bad Effects of Menstruation on a Case of Interstitial, 353
Keratoplasty, Results of, 283
KNAPP and DUANE, Permanent Scotoma Caused by Looking at the Sun

during an Eclipse, and Complicated by Uniocular, Revolving Hemianopsia, 104
KNIES, The Anterior Outflow Channels of the Eye, and the Artificial Production of Glaucoma, 169

"La Clinique Ophthalmologique," Notice of, 142
Lachrymal Apparatus, Report on, 118, 274, 420, 524
Lachrymal Gland, Case of Tumor of the, 275 ; Secretory Nerves of the, 269
Lachrymal Sac, Congenital Monolateral Fistula of the, 251 ; Death Following Probing of the, 420
LANDESBERG, Obituary Notice of, 444
Lens, On the Star-Figure of Man and the Vertebrates, 154 ; Report on the, 128, 288, 432, 534
Lids, Report on Diseases of the, 118, 273, 418, 522
Literature, Report on General Ophthalmological, 109, 263, 410, 514

Macula, Observations on the, 301
MAUTHNER, LUDWIG, Obituary Notice of, 143
MAUTHNER, "Farbenlehre" (The Theory of Colors), Notice of, 141
Measles, Blindness from, 437
Metastatic Ophthalmia, Cases of, 297
Micropthalmus, with Orbito-Palpebral Cyst, 73
MILLIKIN, A Case of Dermoid Cyst of the Orbit, 356
Miscellaneous Notes, 139
MITVALSKY, A Tumor of the Ciliary Body, 161 ; Choroidal Tumors, 450 ; Contributions to the Congenital Anomalies of the Fundus Oculi, 192
Monocular Diplopia, 548
MOULTON, H., Traumatic Paralysis of the Abducens, 363
Muscles, New Suture in Advancement of the, 375
Muscles and Nerves, Report on, 118, 275, 421, 524
Myopia Discission of the Lens in, 272, 417, 499

NAGEL, Obituary Notice of, 556
Night Blindness, and Pigmentary Degeneration, 313
NIMIER and DESPAGNET, "Traité Elementaire d'Ophthalmologie,' Notice of, 140

Ocular Disturbances in General Affections, Report on, 136, 295, 440, 545.
Œdema of the Eyelids, Curious Case of, 85
OELLER, "Ophthalmoscopic Atlas,' Notice of, 139
Ophthalmometer, Improvements of Javal's, 340; How the, can be Utilized into a Chromatometer, 349
Optic Nerve, A Case of Second Attack of Papillitis in Post-Neuritic Atrophy of the, 252 ; Alveolar Fibro-Sarcoma of the, 224 ; Disease of the, and Oxycephaly, 55 ; Hyaline Bodies on the, 395 ; Report on, 132, 293, 438, 542
Orbicularis Pupillary Reaction, 402
Orbit, Dermoid Cyst of the, 356 ; Empyæma of, from Phosphorus Maxillary-Necrosis, 85
Orbit and Neighboring Cavities, Report on, 121, 277, 422, 526
Oxycephaly and Diseases of the Optic Nerve, 55

Paralysis of all Ocular Muscles after Injury to the Skull, 276
Pathology, Diagnosis, and Therapy, Report on, 110, 265, 411, 516
"Perimetry," by Dr. Ole Bull, Notice of, 549
Physiology, Report on, 115, 268, 415, 519
Potash, Permagnate of, 413
Prize Essay, 143

Refraction and Accommodation, Report on Anomalies of, 116, 271, 417, 521

Report on Progress of Ophthalmology in the Second, Third, and Fourth Quarters of the Year 1894, and the First Quarter of 1895, 109, 263, 410, 520
Report of the Transactions of the Twenty-fourth Ophthalmological Congress, Held at Heidelberg, August, 1895.
Retina, Deutschmann's Method of Operating for Detachment of, 538; Histological Alterations in the, after Experimental Injuries, 455; Probable Hemorrhagic Origin of Striæ of the, 147
Retina and Functional Disturbances, Report on, 129, 290, 434, 537
Retinitis Albuminurica, Prognosis *quoad Vitam* when Observed in Bright's Disease, 291, 540
Retinitis Diabetica, 206.
ROOSA, "A Clinical Manual of Diseases of the Eye, Including a Sketch of its Anatomy," Notice of, 139

SCHWEIGGER, C., The Results of the Operation for Strabismus, 8; "Vorlesungen ueber den Gebrauch des Augenspiegels" (Lectures on the Use of the Ophthalmoscope), Notice of, 551
DE SCHWEINITZ, A Second Attack of Papillitis Occurring in a Case of Post-Neuritic Atrophy of the Optic Nerves, 252
Scotoma, Permanent, caused by Looking at the Sun during an Eclipse, 104
Sinus, Osteoma of the Frontal, 423
Society Meetings, 144
Sodium Salicylate, Subconjunctival Injection of, 428
SPALDING, Clinical Contributions, 85
Spring Catarrh, Palpebral Form of, 466
Strabismus, Advancement in all Cases of, 275; Cases of Divergent, with Homonymous Diplopia, 277; Results of the Operation for, 8; Theories of, 1
Sympathetic Ophthalmia, Report on, 287, 432, 502, 533
Syphilis in the Eye, Treated with Mercury, Potassium Iodide, and Pilocarpine Combined, 407
Syphilis, Ocular Affections in, of the Brain, 92, 298

TEPLJASCHIN, Contribution to the Knowledge of the Histological Alterations in the Retina after Experimental Injuries, 455
Tetanus, after Injury to an Eye, 440
Trachoma, Pathological Anatomy of, 282; Treatment of, 281
Tuberculosis, of Eye, 43, 285; of Lachrymal Gland, 76

VEASEY, A Case of Double Coloboma of the Choroid, 202
VIALET, Obituary Notice of, 556
Vitreous, Report on Diseases of the, 288, 434

WALTER, An Itinerant Cataract Operator at the End of the Nineteenth Century, 484
WEILAND, A Straight Arm, Corrected Scale, and Other Improvements for Javal's Ophthalmometer, 340; How Javal's Ophthalmometer can be Utilized for a Chromatometer, 349
WEISS and BRUGGER, Oxycephaly and Disease of the Optic Nerve, 55
WILLIAMS, HENRY W., Obituary Notice of, 555
WINTERSTEINER, A Contribution to the Occurrence and Origin of Congenital Anomalies of the Eyes, 197
Worms, Loss of Vision from the Intestinal Presence of, 296

Yellow Oxide of Mercury Ointment as Influenced by Light, 486

ZENTMAYER and POSEY, One Hundred and Seventy-five Cases of Glaucoma Simplex, 378
ZIMMERMANN, Ocular Affections in Syphilis of the Brain, 92; Traumatic Paralysis of the Abducens Nerve, 243

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